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PERFORMANCE CHARACTERISTICS OF ELECTRICAL HEARING AIDS FOR THE DEAF

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There has long existed a need, which has been expressed by otologists and others interested in persons who are hard of hearing, for a disinterested comparison of the performance characteristics of portable commercial carbon microphone hearing aids. In 1934 Dr. Horace Newhart, as chairman of the Committee of Deafness Prevention and Amelioration of the American Academy of Ophthalmology and Otolaryngology, proposed that an investigation be undertaken to determine whether significant differences exist between the hearing aids of various manufacturers. With the help of funds supplied by the Graduate School Research Committee of the University of Minnesota the proposed investigation was commenced in July 1934 as a joint project of the Division of Otolaryngology and of the Electrical Engineering Department.

It was the original plan to test the products of the leading and representative manufacturers of portable carbon microphone hearing aids and thereupon to make an attempt to specify easily applied procedures for evaluating their relative worth. Accordingly, seven different stock hearing aid models of the portable carbon microphone type were tested in an extensive preliminary investigation. These tests indicated the nature of the data needed to determine the performance characteristics of the instruments and showed which models were comparable as to amplifying power. A second more extensive series of tests was conducted on the latest product of four of the leading manufacturers. This article is a report on this second series of tests, with an introductory discussion of the general problem.

Two general types of hearing aids are in current use, the air conduction type, which is fundamentally similar to the telephone, and the so-called bone conduction type. The first part of the discussion will be confined to the air conduction method of ameliorating deafness.

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It is generally assumed that the measure of a person's loss of hearing is known when the departure of his threshold of hearing for pure tones from the normal threshold is known. As it is impossible to make measurements at each frequency in the range of human hearing, since the number of pitches is unlimited, it is customary to test the subject's hearing acuity with a finite and usually a relatively small number of pure tones. Tests of loss of hearing made with a noise composed of many frequency components are also employed but for the present purpose are not of interest. The methods employed in mapping a person's threshold of hearing and the precautions necessary in securing reliable audiograms have been described by Harvey Fletcher,[1] Vern O. Knudsen and I. H. Jones [2] and others.

In addition to its known value to the otologist in following the progress of deafness in a patient, the audiogram has been assumed to be of value in prescribing a suitable sound-amplifying device. The latter assumption is worth examining closely. It implies that the amount of the amplification which should be supplied to the person suffering from loss of hearing should vary with the frequency,[3] the variations being in some way related to the variation with frequency of the patient's displaced threshold of hearing. A common belief is that the amount of amplification supplied for every frequency interval should be fixed and such as to restore the person's threshold of hearing to normal throughout the audible range. A little reflection will serve to show that this is not a necessary conclusion. It is possible, for example, that a person may not be able to hear faint sounds but may be able to hear speech at the level of loudness of ordinary conversation as well as a person having normal hearing. Such a person when listening to speech of ordinary loudness needs no amplification. What he requires is amplification varying not with frequency but with loudness and varying so as to be great for sounds of low intensities and falling off as the loudness increased, acting much after the manner of the automatic volume control on the modern radio set. The fact that but a small part of the listening that a normal person is called on to do is carried on at the threshold of hearing should serve as a caution in accepting a theory which places the

1. Fletcher, Harvey: Speech and Hearing, New York, D. Van Nostrand Company, Inc., 1929.

2. Knudsen, Vern O., and Jones, I. H.: Laryngoscope **45**:48 (Jan.) 1935.

3. A study of selective amplification under the direction of Vern O. Knudsen is reported to be under way at the University of California at Los Angeles. In a preliminary report made before the Acoustical Society of America on Dec. 6, 1935, Knudsen indicated that selective amplification was found to be somewhat better than uniform amplification. Further study to determine the effect of accommodation is being made.

whole responsibility for loss of hearing on displacement of the threshold of hearing. This suggestion has also been advanced recently by Steinberg.[4]

It would appear that before intelligent prescription of sound amplification can be made for a person with defective hearing it is necessary to know not only the sound intensities at the threshold of hearing but also the equal loudness contours at intervals extending from the threshold of hearing to the loudest tolerable sound levels. Curves of this kind for persons of normal hearing have been determined only comparatively recently. The difficulties of making studies of equal loudness contours for a person who is hard of hearing are considerable, and the ideal which demands that a hearing aid should be prescribed after a test, much as glasses are fitted to a person with defective vision, will no doubt prove to be a matter of much greater difficulty to put into practice if it is to be carried out as has been suggested. It is also obvious that a complete map of a person's hearing perception will be of little use if suitably diverse or adjustable hearing aids are not available.

Until recently it has been generally assumed that a stock hearing aid would serve best if it provided amplification of sound which was reasonably uniform, and manufacturers attempted to produce such an instrument. Suggestions that frequency response curves be furnished with each instrument, however, have not generally been followed, although representative response curves have been published in journals accessible to otologists.[5] Some manufacturers advise the prospective purchaser to make a practical trial of the hearing aid, supplemented by the advice in some cases that a sentence intelligibility test be performed at home. As matters stand, probably no better advice can be given.

Because of lack of sufficiently fundamental knowledge about defective hearing it appears that no entirely rational program of prescribing amplification can be formulated at this time. And if this difficulty were overcome there still remains the question whether in the present state of the art of electric amplification of sound the indicated specifications could be met. If no limitations as to weight, bulk or cost were set, possibly there would be no difficulty. But since an acceptable portable hearing aid must be very light, small and inconspicuous, the modern advances in vacuum tube technic involving the use of the vacuum tube and the acoustic apparatus adapted thereto are not available. At present, reliance must still be placed on carbon particle microphones and carbon particle electrical amplifiers. With these restrictions, it apparently

4. Steinberg, John C.: Telephone Research and Problems of the Deafened, Bell Telephone Quart. January 1936, vol. 15.

5 Fletcher, Harvey: Hopeful Trends in the Testing of Hearing and in the Prescribing of Hearing Aids, Proc. Am. Federation Organ. for Hard of Hearing, Inc. **15**:116, 1934.

requires careful design to meet certain minimum requirements which would appear to be inescapable and independent of any theory. The following would probably be included in any list of such requirements: (1) sufficient amplification of sound free from sharp peaks for response and covering a minimum frequency range of from 200 to 3,000 cycles, (2) freedom from nonlinear distortion noises and from internal noises and (3) freedom from erratic behavior. While these factors are listed as basic, in the end it is only as they affect the ability to hear and interpret speech correctly with a minimum of effort and nervous strain that they are important. It cannot be questioned that the principal reason for seeking mechanical aid is not to hear music or natural sounds but to enable a person to maintain normal contact with his fellows through speech. It follows, therefore, that the crucial test of any hearing aid should depend on this property. Interpretability, then, may be termed the useful output of the device. Outputs of noise or sharp peaks of amplification, on the other hand, while they may appear to increase the over-all amplification, do not contribute to the useful output but, on the contrary, detract from it.

Accordingly, it was decided to rate the hearing aids to be tested basically on their ability to transmit speech sounds and sentences, and secondarily on the prominence or freedom from the defects mentioned. Tests of articulation of sound and of sentence intelligibility were made, based on the technic described by Fletcher and Steinberg.[6]

GENERAL PLAN OF TESTS

The test plan decided on was to place the microphones of the hearing aids in a well damped acoustic test chamber and produce therein, by means of an artificial voice, single frequency tones or speech sounds of known intensity, the intensity to be measured independently by a standard microphone placed near the carbon microphone tested. The acoustic output of the hearing aid was then to be submitted to an artificial ear the electrical output of which could be observed with an oscillograph and compared with the electrical output of the standard microphone within the chamber, or both electrical outputs could be converted to the acoustic form and alternately transmitted to observers for judgment as to loudness, articulation and intelligibility. The artificial voice could be supplied with electrical energy either by a beat frequency audio-oscillator or by voice currents produced by members of the test crews speaking over a high grade transmission system.

It might be thought that the test plan is unnecessarily circuitous and complicated, but reflection will prove the necessity of some such plan. In the first place, persons with normal hearing must be relied on for the tests of articulation. But if the hearing aid is to be operated under normal conditions it may be expected to produce much higher intensities of sound than a normal ear can endure or rightly judge. Accordingly, a uniform loss must be introduced between the receiver of the hearing aid and the human ear. At present the electrical method of producing this loss is the only one available. Likewise, an artificial voice is necessary if

6. Fletcher, H., and Steinberg, J. C.: Articulation Testing Methods, Bell Syst. Tech. J. October 1929, p. 806.

measurements of response to single frequency tones are to be made, since the power of producing pure tones of uniform intensity and variable frequency is beyond human capability. These and other points were discussed by Inglis, Gray and Jenkins.[7]

Test Apparatus.—Figure 1 shows in diagrammatic form the various units of the test arrangement, with descriptive designations. The acoustic chamber was constructed of wood framing lined with acoustic celotex 1¼ inches (3.2 cm.) thick and further damped by balsam wool blanket[7a] to a depth of 6 inches (15 cm.) at both ends. By moving the standard microphone within the chamber and observing its output it was verified that the chamber was substantially free from variations in pressure due to reflections for all frequencies above 200 cycles per second. For lower frequencies the variation was not of much importance, as it was found that the response of all the hearing aids tested was practically zero for these frequencies, and in any event the effects of reflection with low frequencies were tolerable.

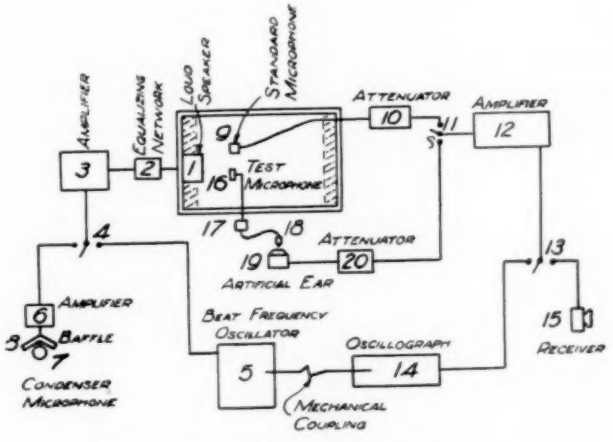

Fig. 1.—Diagram of the test apparatus.

Sound was produced in the chamber by a 7 inch (17.8 cm.) loud-speaker of the cone type (*1*) supplied through an equalizing network (*2*) and an amplifier (*3*) via a switch (*4*), either with pure tones by a beat frequency oscillator (*5*) or with voice currents from an amplifier (*6*) and a condenser microphone (*7*) located in an adjoining room. When tested for frequency response, with the terminal voltage of the oscillator (*5*) remaining constant and a standard microphone (*9*) placed 18 inches (46 cm.) from the loud-speaker (*1*), it was found that the output of the microphone as measured at the terminals of the amplifier (*12*) with an oscillograph (*14*) was constant to within 9 decibels in the range of from 200 to 3,900 cycles. An oscillographic record of the variation in pressure with the frequency is shown in figure 7. This record was made by mechanically belting the drum of the oscillograph and the dial of the beat frequency oscillator together and

7. Inglis, Gray and Jenkins: A Voice and Ear for Telephone Measurements, Bell Syst. Tech. J. **11**:293, 1932.

7a. A commercial sound-absorbing material.

rotating the dial manually, pausing at 200, 500, 1,000, 2,000 and 3,000 cycles to produce the marker lines for frequency. While the measuring apparatus itself is not wholly free from error, it nevertheless is satisfactory for the purpose, since all the hearing aids tested exhibited much greater variations. Indeed, all the hearing aids tested varied so greatly in amplification that it was difficult to obtain satisfactory oscillographic records with a linear recording device.

Calibration of Test Apparatus.—The transmission link starting from the microphone (*9*) to the Western Electric type 555 moving coil type of receiver (*15*) was first adjusted so as to transmit speech sounds without gain or loss. This was done by placing the receiver (*15*) and an observer within the acoustic chamber in such a way that the observer could alternately listen with both ears to the sound proceeding from the loud-speaker (*1*) and then with one ear to the sound proceeding from the receiver (*15*). The intensity of sound was adjusted so as to be at the level judged to be that of normal speech. In the first instance the observer faced the sound, keeping both ears the same distance from the loud-speaker as was the microphone (*9*), and in the second case the free ear was tightly covered with a water tumbler stuffed with wool yarn. With the help of a person stationed at the attenuator (*10*) this transmission link was adjusted until the two sounds appeared to be equally loud. Three observers agreed on this adjustment to within 1 decibel, and the average value so determined was kept fixed throughout the subsequent measurements.

Single frequency checks were also made on the adjustment, which agreed within 2 decibels in the range of from 500 to 4,000 cycles with the value determined by calibration with speech. Strictly considered, it is possible to adjust a system of this kind to equality only when the sense impressions in the two cases are identical, so that for the case in question it would be necessary to have the two sound-producing devices, the loud-speaker (*1*) and the receiver (*15*), identical and to listen to their respective outputs in exactly the same way. However, it is inherent in the nature of the information sought, namely, the amplifying power of hearing aids, that the sound is impressed on the microphone in a different way than on the user's ear. This circumstance requires the employment of some such method of adjustment as that adopted. It was found by trial that the sense impressions received in the two cases were similar and that the attenuator settings were reproducible.

Next the transmission link starting from the artificial ear (*19*) to the receiver (*15*) was adjusted so as to transmit speech sounds without gain or loss. By supplying one of the receivers of a hearing aid device with voice-modulated current from the output of the amplifier (*3*) and alternately listening to the output of the receiver directly and then with the transmission link inserted, attenuator *20* was adjusted for equality of loudness. Two observers agreed closely on the setting adopted. Single frequency observations checked with the speech calibration satisfactorily except for 2,500 cycles, at which 9 decibels greater attenuation was found to be necessary. The quality of speech transmitted over the link, however, was not perceptibly altered, and the difference was allowed to stand. Two types of receivers such as are commonly supplied with hearing aid sets were used in this calibration, the watch-case type and the small, ear-fitted type. In this adjustment the two yielded identical attenuator settings. The calibration of attenuator *20* determined in this way was noted and hereafter will be referred to as the zero setting. To obtain the gain of the various hearing aids, speech was transmitted over the system from *7* to *15*, and by operating the switch (*11*) the standard link or the link containing the hearing aid was connected in the circuit alternately. Attenuator *20* was adjusted until the observer at *15* indicated, over a simple signaling system,

that the loudness as transmitted over the two links was the same. The attenuation in *20* over and above the zero setting then indicated the gain of the hearing aid.

Some care was necessary in comparing the levels of loudness of the standard with those of some of the hearing aids because of the great difference in quality. However, agreement in the results obtained by different observers was fairly close, even in these cases. In judging equality of loudness, observers were instructed to disregard differences in intelligibility. This must, of course, be remembered in the final comparison of the four hearing aids. It will be observed that because of the method of calibration the intensity of sound at the observer's ear was always that which was originally judged to be the average listening intensity for normal speech and hence was the optimum intensity for the exercise of the observer's acoustic judgment.

For testing articulation, consonant-vowel-consonant syllables were constructed as described by Fletcher and Steinberg,[6] and likewise tests of sentence intelligibility were carried out as described by these authors.

The test crews were selected, after preliminary audiometric examination to establish normalcy of hearing, principally from a group of students, male and female, enrolled in classes for the study of speech at the University of Minnesota. Periods of reading and listening for each person were limited to about fifteen minutes at each session.

The microphone before which the reading was done was located in a small empty classroom having good acoustic properties. At some distance behind the microphone was placed a celotex absorbing baffle (*8*), the position of which was determined by trial and by the concurrence of three observers at the receiving end of the high grade transmission system as to the most natural hearing conditions.

HEARING AIDS TESTED

The four hearing aid models tested will be referred to by the numbers I, II, III and IV. All four models were equipped with a carbon microphone transmitter, a battery, a gain control rheostat, a carbon microphone power amplifier, one molded ear-fitting small receiver and one bone conduction oscillator. In addition, unit I was furnished with a large watch-case type of receiver, and unit IV was equipped with three small receivers, intended for low, medium and high frequency ranges and accordingly marked *L*, *M* and *H*, respectively, and also three bone conduction oscillators similarly marked.

MEASURED SPEECH AMPLIFICATION

The amplification obviously varies with the setting of the gain control rheostat of the hearing aid. Since the largest amplification may be attended by undesirable effects, it was decided to measure amplification for other rheostat settings also. The table gives measured amplifications in decibels for full range rheostat settings and for half range rheostat settings. The current supplying each unit was taken from the same set of three new dry cells. The tabulated values are the averages of three or more observers.

UNIFORMITY AND RANGE OF RESPONSE

None of the units tested had even an approximately uniform frequency response characteristic. Consequently it is concluded that whatever may be the differences in performance they are probably not chargeable to a difference in uniformity of response. Several oscillograms for each of the four units for various rheostat settings were obtained by the same method as that described in connection with figure 7. The various ways in which the hearing aids tested differed in nonuniformity, however, are not considered significant or worth exhibiting. This will be made clearer later.

Speech Amplification for the Four Units, in Decibels

Unit I	Unit II	Unit III	Unit IV
Full Range Rheostat Settings			
26 (small receiver)	41	30.5	20 (receiver H) 20 (receiver M)
31 (large receiver)	..	....	25 (receiver L)
Half Range Rheostat Settings			
23 (small receiver)	23	27.0	18 (receiver H) 18 (receiver M)
27 (large receiver)	..	....	20 (receiver L)

It should be understood that it is contended not that uniformity of response is not important or desirable but that none of the units so far tested has this quality. And since the units differ greatly in their ability to transmit speech, it appears that one must look elsewhere for causes of differences in performance. It is known, for example, that the frequency range of a transmission system is important. One of the seven hearing aids tested in the preliminary investigation was found to cut off all frequencies above 1,800 cycles per second and on being tested showed a correspondingly poor capacity for transmission of speech.

The four hearing aids reported on in this article were approximately similar in frequency range. None of them responded perceptibly below 200 cycles; the response was small up to 500 cycles, and all had a fairly sharp upper frequency cut-off in the neighborhood of 3,000 cycles. Unit III was exceptional in having a somewhat higher upper frequency limit with a small output as high as 4,000 cycles.

INTELLIGIBILITY AND ARTICULATION

None of the measurements thus far cited discriminated greatly between the four units tested. The results of measurements of intelligibility of discrete sentences and of articulation of vowels and consonants, however, yielded decisive information. Figure 2 shows the averaged results when the hearing aids were used with the maximum possible

amplification, and figure 3 shows corresponding values for half range rheostat settings. In comparing the data on amplification, what is important is the actual measured amplification, not the rheostat setting. This amplification in decibels is shown near the base of each column, which by its height indicates the percentage of intelligibility, that is, the percentage of the sentences heard which were correctly interpreted.

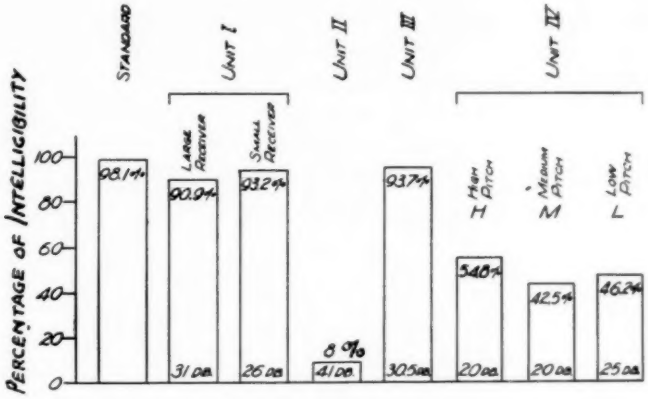

Fig. 2.—Percentage of intelligibility with full range rheostat setting.

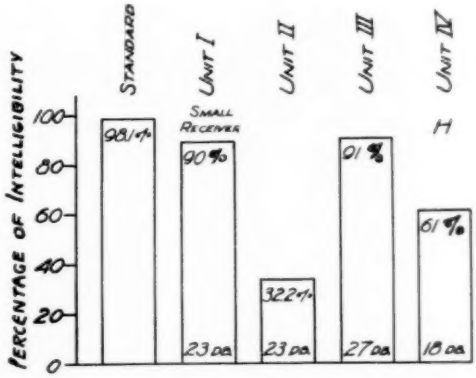

Fig. 3.—Percentage of intelligibility with half range rheostat setting.

It will be observed that units I and III are vastly superior to units II and IV. Unit IV shows up poorly not only in the percentage of intelligibility but also with respect to amplification. The different-pitched receivers, indicated by *H*, *M* and *L*, with which unit IV was equipped apparently had little effect on either intelligibility or loudness. The poorest showing as regards intelligibility was made by unit II, which at the same time had the highest observed amplification, 41 decibels. Apparently this high gain was due to a great extent to distor-

tion noise components and should in reality be regarded largely as loss. When the performance of the four units at half rheostat range setting is analyzed it will be seen that the apparent amplification of unit II was reduced by the largest amount and compares reasonably well with the others in this respect and also that the percentage of intelligibility increased greatly but still remains the poorest.

The intelligibility of the standard and practically distortionless reference system was found to be 98.1 per cent. The best of the hearing aids, units I and III, compared favorably (with an intelligibility of 93.2 and 93.7 per cent, respectively) with the distortionless standard, in spite of the fact that a great difference in the quality of speech was observable.

Tests of articulation of sounds served further to differentiate between the hearing aid units. In figure 4 are shown the results of tests

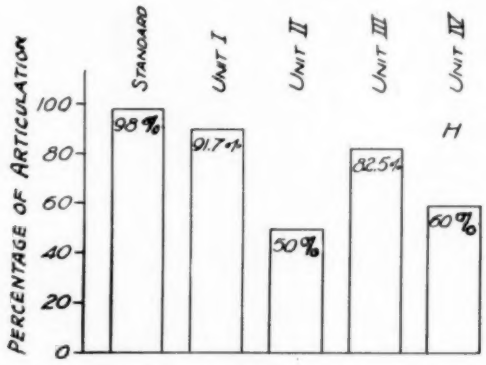

Fig. 4.—Percentage of vowel articulation with half range rheostat setting.

of articulation of vowels and in figure 5 the corresponding results for articulation of consonants. These may be described briefly as the sounds correctly heard in terms of percentage of the total number of consonants or vowels contained by meaningless monosyllables transmitted by the unit tested. The results shown in figures 4 and 5 are for conditions of transmission identical with those under which the values shown in figure 3 were obtained. The corresponding results for the full range rheostat setting are not given for the reason, as has already been noted, that unit II was in this condition apparently greatly overloaded and comparison is meaningless.

With reference to figures 4 and 5, it will be observed that hearing aid units I and III are greatly superior to units II and IV. They compare favorably with the standard system as regards articulation of vowels but are considerably poorer than the standard in their ability to transmit consonants without distortion.

Everything considered, it appears that unit III is the best of the four, since, when it is compared with unit I, which is equipped likewise with a small ear-fitting type of receiver, it has an amplification greater by 4.5 decibels and has approximately the same rating for intelligibility and for articulation of sounds. Both unit I and unit III, however, are greatly superior in every desirable quality to unit II and unit IV.

In view of the fact that the four hearing aids differ so little comparatively in uniformity of response, frequency range and amplification, it is natural to inquire why they differ so greatly in the rating for intelligibility and for articulation. It was suspected that the difference was due to nonlinear distortion, particularly because of the behavior of unit II, as shown on figures 2 and 3. For it will be observed that as the apparent amplification was decreased the percentage of intelligibility increased—a circumstance easily explained on the assumption that an

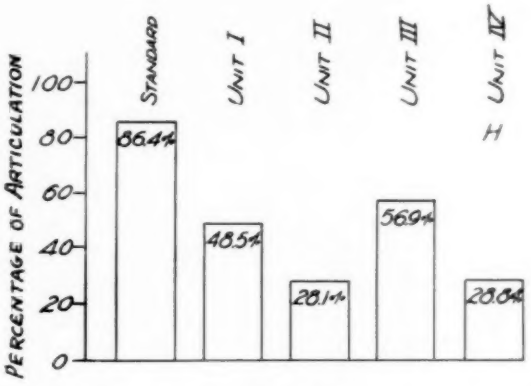

Fig. 5.—Percentage of consonant articulation with half range rheostat setting.

increasingly large part of the sound produced in the receiver with increase in amplification is due to noise components. These noise components may be harmonic or inharmonic amplified components of the speech sounds; in any event, they serve to mask the latter and hence decrease the intelligibility.

Direct evidence of the presence of nonlinear distortion was obtained by exposing the microphones of the hearing aids to single frequency tones in the acoustic chamber of figure 1 and observing the output of amplifier 12 by means of a cathode ray oscilloscope. Some of the patterns observed were photographed and are shown in figure 6.

Many of the observations made by this method were not photographically reproducible and can only be described. All the four hearing aids showed nonlinear distortion effects at certain frequencies, but units I and III were free from them except in a few narrow frequency bands,

whereas units II and IV showed bad distortion except in a few narrow bands. The behavior of all the units was more or less unstable, so that successive images formed on the screen of the oscillograph did not coincide, as they did, for example, when the standard microphone link was substituted for that containing the hearing aid. This instability was particularly poor in units II and IV, especially in regions of poor wave form. On the other hand, unit III was almost free from this defect, and unit I was second best. Although the method is unsuited for quantitative analysis, the conclusion was unmistakable that the hearing aid units which had a low intelligibility rating also produced the greatest amount of observable distortion of wave form and were most unstable.

By means of another method, now to be described, further information concerning distortion was obtained. The method consists of pro-

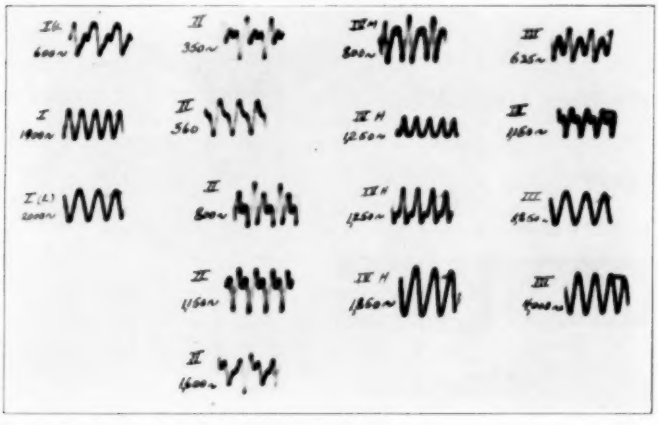

Fig. 6.—Oscillograms showing comparative nonlinear distortion effects of the four hearing aid units at various frequencies.

ducing two pure tones in the acoustic chamber, one fixed in frequency and the other varied manually from 200 to 3,900 cycles. Low pass filters inserted ahead of the oscillograph (*14*, fig. 1) permit only frequencies below the cut-off of the filter to register on the oscillograph. The oscillograph is so coupled to the oscillator (*5*) that as the oscillator frequency is varied from 200 to 3,900 cycles the film drum is rotated through one revolution. In consequence, any modulation products of frequency sufficiently low to pass the filter which are produced by the double tone excitation of the hearing aid will be registered by the oscillograph.

Some of the modulation oscillograms obtained are shown in figure 7, the intensities of sound used in each case being no greater than normal intensities of speech. Each trace is marked with the number of the film, the code number of the hearing aid, the cut-off frequency of the filter

(f_c) and the fixed frequency tone (f_o). In film 47-2 the response of the standard microphone and amplifier is shown; this will be observed to be free from nonlinear modulation effects. Immediately below is a similar oscillogram for unit II for half range rheostat setting. The effect in this case of the variable frequency excitation on the response to the steady 1,000 cycle tone was marked. By listening to the output the presence of strong extraneous frequencies was verified. In the next oscillogram (51-4) the cut-off frequency of the filter ($f_c = 600$) was less than the fixed frequency ($f_o = 1{,}600$), and in consequence

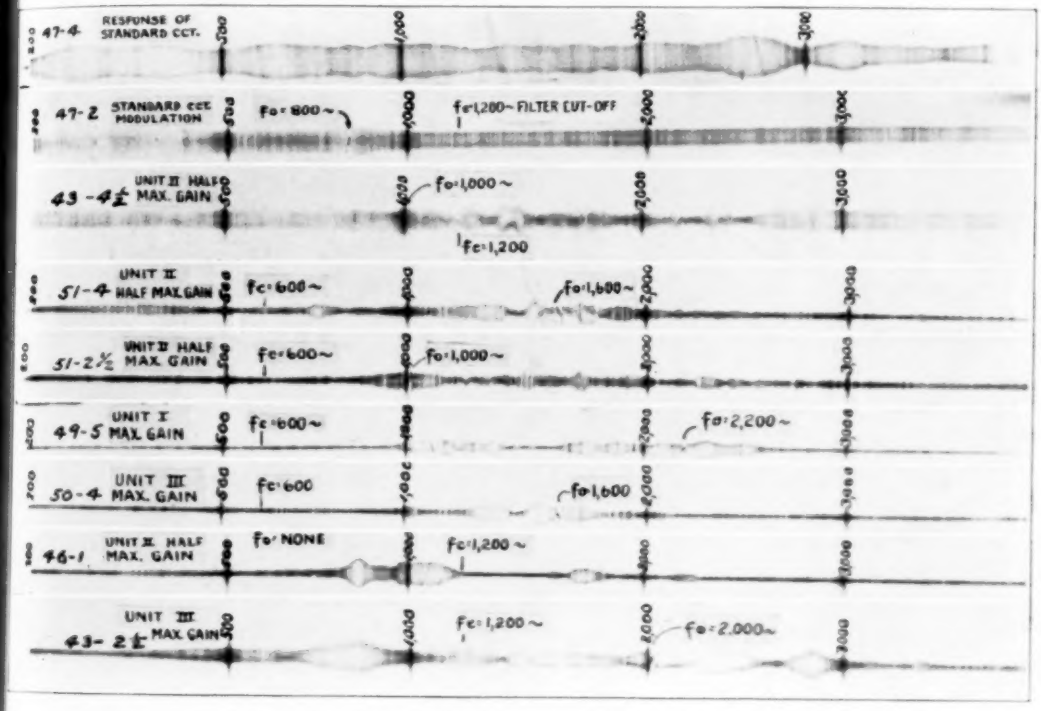

Fig. 7.—Oscillograms for hearing aid units, taken with air conduction oscillators.

all the output shown above the 600 cycle mark on the oscillogram is in reality a low frequency output due to modulation. It will be observed that these distortion components are greater in amplitude than the combined exciting outputs of tone observable below the 600 cycle mark. This is to be compared with the behavior of the standard circuit for proper judgment.

The remaining oscillograms show similar effects observed for the four units. In comparing the hearing aid units it should be remembered that the oscillograms of figure 7 exhibit only a small part of the distortion characteristic. To be complete the fixed frequency should take on

values covering the total range of response of the instruments, and in addition the entire series of recordings should be repeated, high pass filters being used to permit the high frequency distortion components to register.

Obviously, such evidence would be excessively abundant, and instead of it is offered a rough comparison of the four units. By observation the fixed frequency (f_o) can be set successively at the points producing the greatest amount of distortion by interaction with the variable frequency. When this was done for all four hearing aids, it was found, as the oscillograms show, that all produced distortion. Relatively, unit II was the worst by a large margin in this respect, even when the gain control rheostat was set at half maximum amplification. Unit IV had an intermediate rank, and units I and II were most free from this kind of distortion.

BONE CONDUCTION OSCILLATORS

It is not proposed to enter into an examination of the exact mode of operation of the bone conduction method of affecting the auditory nerve. Békésy [8] appears to have approached the problem in a fundamental fashion. However, no success was had in devising a rational test for the bone conduction oscillator. After preliminary trials it was arbitrarily decided to compare the hearing aid units with the arrangement now to be described.

A piece of leather belting 3/16 inch (0.5 cm.) thick and 3 inches (7.5 cm.) wide was loosely held in a horizontal position between supports about 6 inches (15 cm.) apart. The bone conduction oscillator under observation was adjusted against the lower side of the leather with the spring band supplied by the manufacturer and fastened as closely as possible in the same manner as intended for use on the human head. A phonograph reproducer of the hill and dale type was then laid against the top side of the leather directly over the oscillator. The output of the reproducer was amplified, and oscillographic records of the frequency response were obtained for the four hearing aid oscillators. Samples of these records are shown in figure 8. They are not considered as having great significance in an absolute sense. It is interesting to observe, however, that the departure from uniformity of response for all the bone conductors measured was not as great as that for the corresponding air conduction units. In particular, unit I in figure 8 is seen to be free from excessive variation in the range from 200 to 4,000 cycles. The rather small effect of the low and medium pitch oscillator of unit IV on the vibratory output is also shown.

Measurements of the intelligibility of the bone conduction units were made by converting the electrical output of the phonograph reproducer

8. Békésy: Zur Theorie des Hörens bei der Schall aufnahme durch Knochenleitung, Ann. d. Physik [5] 11 Page 227, 1931.

to speech output in the receiver (*15*, fig. 1). These tests did not change the order of merit of the four units as previously determined, but the differences exhibited were not as great.[9]

A SIMPLE TEST PROCEDURE

At the outset one of the declared purposes of the investigation was to specify easily applied procedures for determining the relative worth of competitive hearing aids. None of the tests thus far described satisfy this specification. Indeed, no test at once simple and completely satisfactory was found. The common advice given to a person who is hard of hearing to try various hearing aids is unsatisfactory, particularly when the person is a poor judge even of perfectly transmitted speech because of lack of practice.

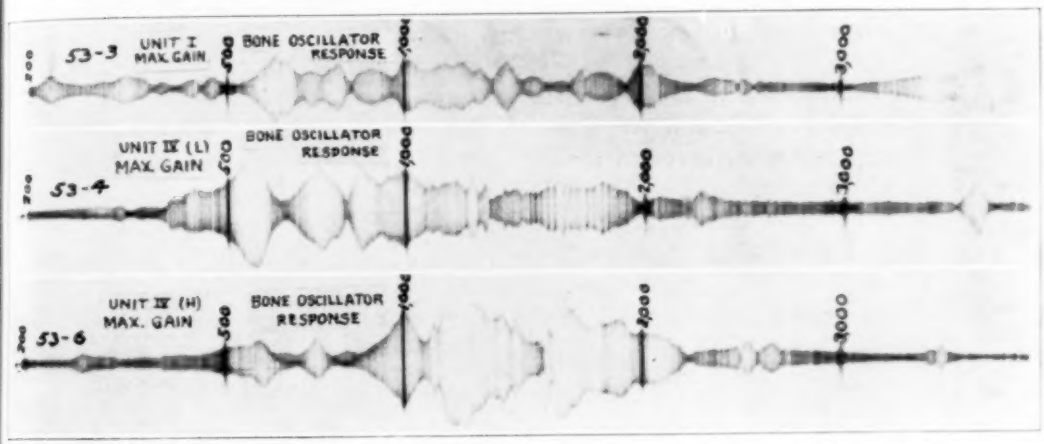

Fig. 8.—Oscillograms for hearing aid units, taken with bone conduction oscillators.

Moreover, it appears that the outstanding defect of portable hearing aids as at present manufactured is a type of distortion which results in the production of extraneous interfering noises. Presumably, persons with normal hearing could detect this defect fully as well as those for whom the hearing aids are designed.

On the basis of these considerations the following test procedure was tried: An exponential horn with an opening of 22 inches (55.8 cm.) was provided at the throat with a tapered fitting so as to match the holes in the receivers of the hearing aids. The microphone of the hearing aid and a speaker were placed in one room, and a number of

9. It should not be inferred from this statement that in clinical cases in which there is increased bone conduction, the bone conduction receiver does not possess great advantages over the air conduction receiver. This aspect of the subject was not considered in our problem.

observers were placed in an adjoining room, with a door between; tests of sentence intelligibility and of syllable articulation were then performed. Although the method is open to the objection that the horn itself produces distortion of amplitude and also that no numerical measure of the amplification is obtained, it nevertheless does permit hearing aids of different performance characteristics to be differentiated. When they differ greatly, as the present commercial devices may be expected to do, the poorer ones may readily be eliminated by this means. The equipment required is such as the otologist can easily obtain and operate.

CONCLUSIONS

Among the various hearing aids of different manufacture on the market having as components a carbon microphone, a carbon amplifier and an ear-fitting receiver, there are important differences in the amount of amplification produced and tremendous differences in the intelligibility ratings.

The differences in performance do not depend measurably on the observed nonuniformity of response. In consequence, furnishing frequency response curves to the otologist with each hearing aid would be of no value.

Of greater importance is a wide range of response (as a minimum, from 200 to 3,000 cycles per second), freedom from singular peaks of amplification and sufficient amplification.

Of paramount importance is the actual aid that the user obtains in interpreting speech sounds and sentences correctly.

Hearing aids having a low intelligibility rating were also found to produce the greatest volume of noise by-products due to nonlinear distortion. It is concluded that these noise components are the principal cause of the poor intelligibility.

Portable hearing aids consisting of a carbon microphone and receiver only are largely ineffective because of insufficient amplification.

At present probably the best means of determining the personal usefulness of a carbon microphone hearing aid of known merit for a given person is the discrete sentence intelligibility test.

An easily performed test for eliminating the most inefficient commercial hearing aids from consideration is suggested.

Tests made with bone conduction oscillators indicated that the relative merit is not appreciably affected by the substitution of bone conduction for air conduction when carried out on persons with normal bone conduction.

The Graduate Research Committee of the University of Minnesota supplied funds for needed apparatus and help. Mr. L. E. Norton aided in the investigation, and Mr. Franklin H. Knower, of the Speech Department, helped to organize the articulation test crews.

A CYTOLOGIC STUDY OF THE EFFECTS OF DRUGS ON THE COCHLEA

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SAN FRANCISCO

The effects of various toxic agents such as drugs, poisons and toxins on the mechanism of hearing are more readily studied by tests of hearing than by examination of sections of the cochlea for pathologic changes. The electrical method for testing hearing in animals now makes it possible to correlate the two phases of the work, and a subsequent paper will deal with the electrical response of the cochlea in relation to the cytologic observations reported here.

Wittmaack,[1] Beck [2] and others have described changes in the spiral ganglion cells and peripheral acoustic nerve fibers following experimental drug intoxications in animals. Quinine and sodium salicylate are the principal drugs for which alterations have been observed, although similar changes occur after the administration of methyl or ethyl alcohols in guinea-pigs. Bacterial toxins have also been found to provoke a similar reaction. In spite of these important findings the use of drugs which impair hearing has continued. More recently, the clinical interest in these problems has been revived (Taylor [3]).

The long and tedious methods for the demonstration of pathologic changes in the cochlea and its various structures obscure important effects of drugs. With such methods the early mitochondrial changes within the hair or sensory cells of the organ of Corti are impossible to demonstrate. The present observations deal with cytologic alterations following the administration of quinine or salicylate to animals. For this purpose, it was found necessary to eliminate the step of decalcification from the technical procedure.

EXPERIMENTAL PROCEDURES

Sixteen animals, including five rabbits, six guinea-pigs and five white mice, were given subcutaneous injections of a 2.5 per cent solution of quinine bisulfate

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From the George Williams Hooper Foundation, University of California.

Supported by a grant from the John C. and Edward Coleman Memorial Fund, University of California.

1. Wittmaack, K.: Der Angriffspunkt des Chinins im Nervensystem des Gehörorganes, Arch. f. d. ges. Physiol. **95**:234, 1903.

2. Beck, K.: Experimentelle Untersuchungen über den Einfluss von Bakterientoxinen und -giften aus dem Gehörorgan, Ztschr. f. Ohrenh. **68**:128, 1913.

3. Taylor, H. M.: Prenatal Medication as a Possible Etiologic Factor of Deafness in the New-Born, Arch. Otolaryng. **20**:790 (Dec.) 1934.

(200 mg. per kilogram of body weight) daily over periods varying from two to eight weeks. A similar number of animals were given daily injections of 2.5 per cent solution of sodium salicylate in equal doses and over periods ranging from three to eight weeks. Sixteen animals, including a similar number from each species, served as controls. Approximately one third of the animals were put to death when drug intoxication became apparent. Others were killed as the reaction became more severe.

Each animal was anesthetized and perfused with warm physiologic saline solution and finally with a diluted solution of formaldehyde U.S.P. (1:10). The procedure for sectioning and staining the animal's cochlea was essentially the same as that described by Guild.[4] Sections 10 microns in thickness were made, and each fifth section was mounted in order to make two series of slides, each series representing every tenth section. One series was stained with hematoxylin and eosin, and the other series, with a modified Van Gieson stain.

The guinea-pig was selected as the experimental animal for studying the effects of the drugs on certain cell constituents. It was found that the mitochondrial fixatives, such as Regaud's solution,[4a] sufficiently decalcified the thin bone of the cochlea to permit sectioning of the block in paraffin. While it was sometimes impossible to secure complete sections for orientation of the various structures in the cochlear duct, enough sections were obtained to justify conclusions.

Fifteen guinea-pigs received quinine bisulfate and fourteen received sodium salicylate over a period of from three to twelve weeks in doses similar to those given to the first series of animals. Ten guinea-pigs served as controls. In addition to the animals mentioned, three pregnant guinea-pigs received quinine bisulfate and three received sodium salicylate. All the animals were put to death by decapitation. One cochlea of each animal was dissected free and placed in Regaud's fixative for three days with changes daily, after which it was placed in 2.5 per cent potassium bichromate with changes daily for four days. The other cochlea of each animal was placed in Bensley's A.O.B. mixture [4a] for twenty-four hours. Before fixation, a small opening was made in the apex of the cochlea and also in the round window to permit more rapid penetration. It was found that the hair cells of the organ of Corti were without distortion and vacuolation following fixation in the A.O.B. mixture, but the mitochondria in the cells of the stria vascularis were better preserved after treatment with Regaud's fixative. Likewise, the latter fixative produced good results for the spiral ganglion cells.

OBSERVATIONS

The ears from the animals perfused with fixative, the cochleae of which were subsequently decalcified, sectioned and sections stained, revealed no new information. The cells of the external sulcus were usually swollen and vacuolated. The stria vascularis occasionally appeared hydropic. The spiral ganglion cells, especially in the lower regions of the modiolus, showed a loss of ground substance with varying degrees of chromatolysis. The aforementioned alterations were not always restricted to the basal half of the cochlea; in the group of ani-

4. Guild, S. R.: War Deafness and Its Prevention: Report on the Labyrinths of the Animals Used in Testing of Preventive Measures, J. Lab. & Clin. Med. **4**:153, 1919.

4a. McClung, C. E.: Handbook of Microscopical Technique. New York, Paul Hoeber, Inc., 1929, pp. 201 and 202.

mals which had received the drugs for as long as eight weeks, the apical half often exhibited similar changes. In a few instances the organ of Corti was lacking, but this phenomenon appeared rather as an anomaly than as an effect of the drug, since it was also encountered in control animals.

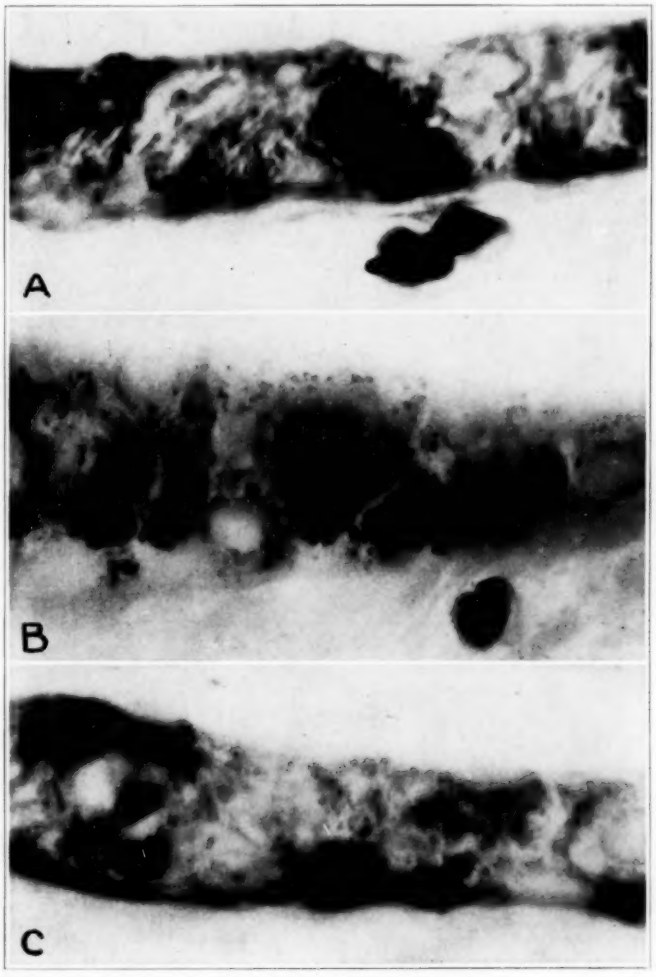

Fig. 1.—*A*, the appearance of the mitochondria of the stria vascularis in a control animal. They are long rods or filaments oriented at right angles to the base of the cell. *B*, the stria vascularis of a guinea-pig which had received subcutaneous injections of a solution of sodium salicylate for four weeks. The mitochondria have lost their original shape and now appear rounded or spherical. *C*, the stria vascularis of a fetal guinea-pig. The pregnant animal had received subcutaneous injections of a solution of quinine bisulfate over a period of three weeks. The mitochondria are drastically altered. Less drastic alterations were apparent in the stria vascularis of the pregnant animal. Regaud fixation; aniline acid fuchsin and methyl green staining; × 1,560.

The series of animals' ears prepared for mitochondrial studies revealed alterations of cell constituents in the different structures of the cochlear duct. The findings for the two drugs were of a similar nature. The histopathologic effects will not be considered under separate head-

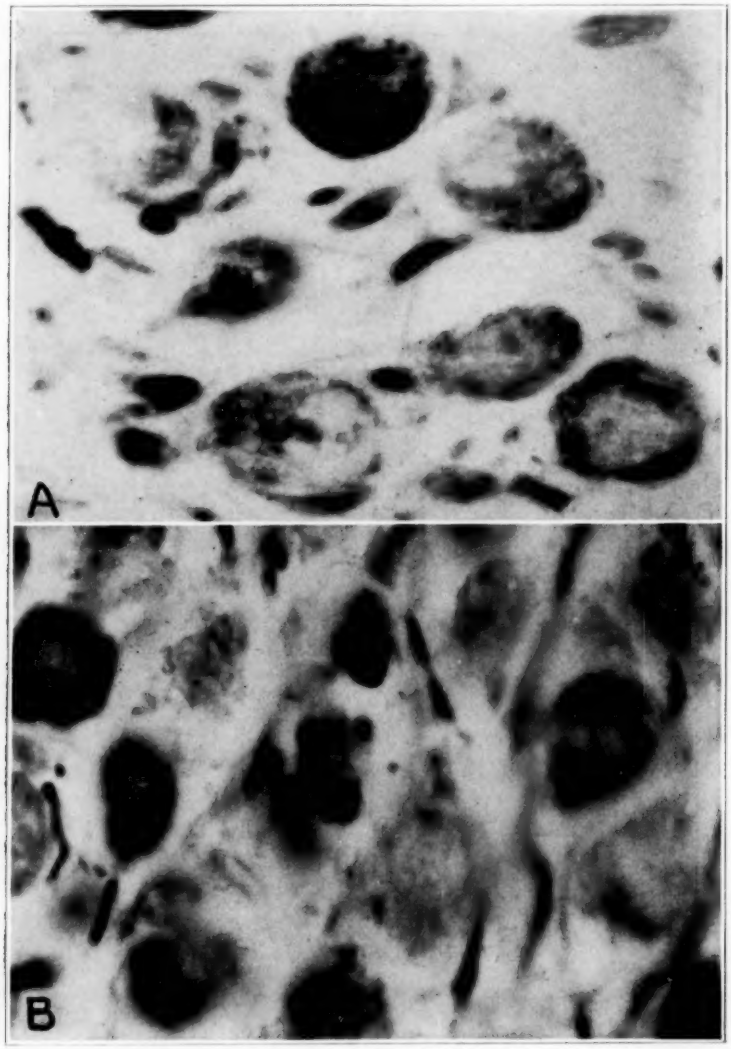

Fig. 2.—*A*, the spiral ganglion cells from a control guinea-pig. Note the appearance of the mitochondria. *B*, the spiral ganglion cells from a guinea-pig which had received subcutaneous injections of a solution of sodium salicylate over a period of eight weeks. The mitochondria retain their normal appearance, but the ground substance of the cells is greatly diminished and the cell outlines are irregular. Regaud fixation; aniline acid fuchsin and methyl green staining; × 1,560.

ings. The dissimilarities of action of the two drugs will be discussed later.

Stria Vascularis.—The mitochondria of the cells of the stria vascularis are long slender rods or filaments and are illustrated in figure 1*A*.

They are closely packed within the cells at right angles to the fixed border and resemble the mitochondria of the convoluted tubules of the kidney. Figure 1*B* serves to illustrate the mitochondria in the basal turn of a cochlea from an animal which had received subcutaneous injections of the solution of sodium salicylate for four weeks. They are no longer rod shaped. They have lost their orientation and now appear spherical. Even more drastic are the mitochondrial alterations within the stria vascularis from an unborn guinea-pig (the pregnant

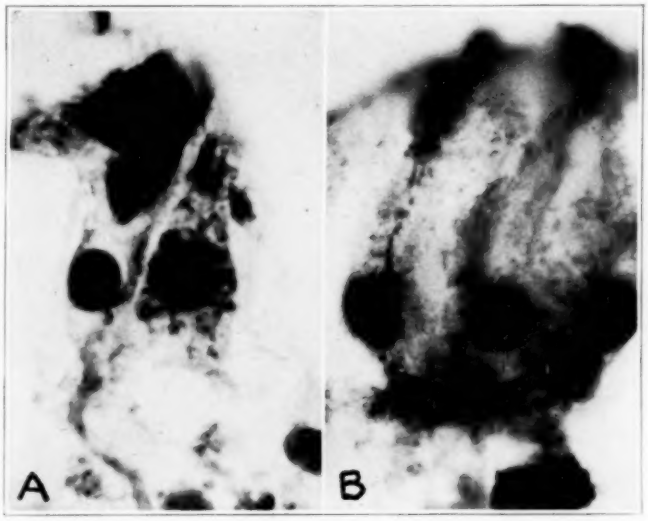

Fig. 3.—*A*, photomicrograph of an internal hair cell from a control animal. *B*, photomicrograph of a group of external hair cells. The cell to the left is in focus and shows the type of mitochondria present. The remaining cells do not reveal their mitochondria because of the plane of focus. A.O.B. fixation; aniline acid fuchsin and methyl green staining; × 1,560.

animal having received the solution of drug subcutaneously for three weeks). The control fetal ears show mitochondria in cells of the stria vascularis similar in number but of less diameter than were those shown in corresponding cells of adult animals. Figures 1*C* and 4*D* serve to illustrate the changes in the form of the mitochondria after administration of quinine. Similar changes were observed in the stria vascularis of fetal ears of animals after injections of the solution of sodium salicylate.

External Sulcus.—The mitochondria of the cells of the external sulcus, unlike those of the stria vascularis, are long slender filaments with no tendency to orient themselves with the border of the cells. Although these cells in the animals which had received drugs were hydropic, the mitochondria remained unaltered.

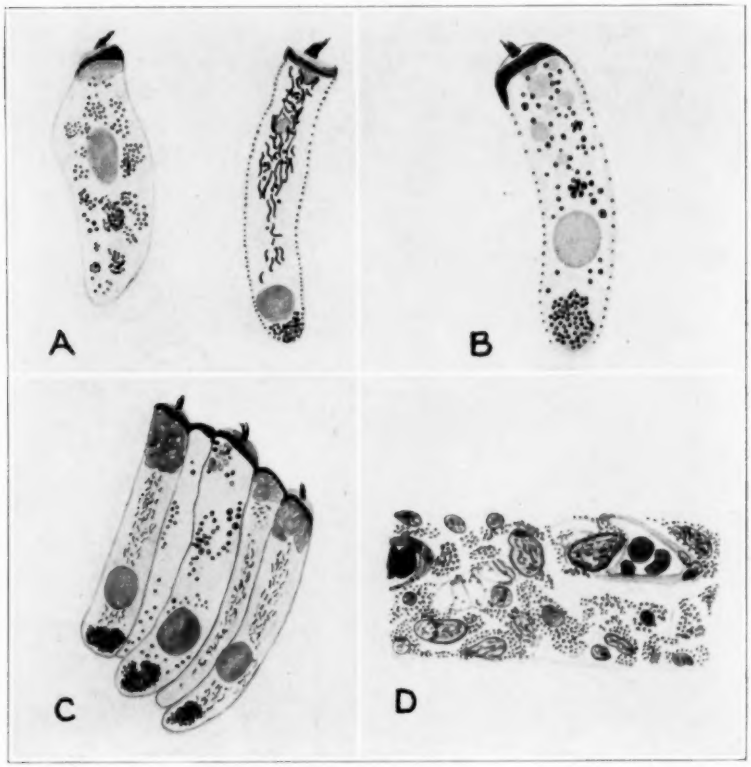

Fig. 4.—*A*, the appearance of the mitochondria in the internal and the external hair cells of the control animals. The internal hair cell reveals mitochondria of a granular type with a tendency to clump. The external hair cell shows slender elongated rods. *B*, an external hair cell from the basal turn of the cochlea of a guinea-pig which had received subcutaneous injections of a solution of quinine bisulfate over a period of four weeks. The cell is wider in diameter than normally. The mitochondria are tiny droplets. *C*, a group of external hair cells from the second turn of the cochlea of a fetal guinea-pig. The pregnant animal had received subcutaneous injections of a solution of sodium salicylate for three weeks. Two cells show alteration of the mitochondria while the adjacent cells do not exhibit this change. *D*, the stria vascularis of a fetal guinea-pig. The pregnant animal received subcutaneous injections of a solution of quinine bisulfate over a period of three weeks. The mitochondria have lost their rod form and are now spherical. Regaud fixation; aniline acid fuchsin and methyl green staining; × 1,000.

Spiral Ganglion.—The mitochondria of the spiral ganglion cells in a control animal are illustrated in figure 2*A*. They are very short rods and similar to those occurring in other nerve cells, such as the spinal ganglion cells. Figure 2*B* illustrates the changes in the appearance of these cells after administration of sodium salicylate for eight weeks. The mitochondria are apparently unaffected by the drug, but there is a loss of ground substance for certain of the cells, and the remaining cytoplasmic substance stains faintly. This condition, although present in the animals which had received quinine, was more pronounced in the group which had received the salicylate.

Organ of Corti.—The normal mitochondrial contents of the internal and external hair cells of the organ of Corti are shown by means of photomicrographs and camera lucida drawings (figs. 3 and 4*A*). The outer hair cells and the inner hair cells differ in the type of mitochondria present. The mitochondria in the outer hair cells are slender delicate rods. Those in the inner hair cells present a granular and clumped appearance. Alterations in the mitochondrial content attributable to drug action are pronounced in the external hair cells. Figure 4*B* illustrates the type of change frequently encountered, usually in the basal turns of the cochlea. The cell is swollen, and the mitochondria have lost their original shape and are rounded or spherical. The group of external hair cells in figure 4*C* are from a lower middle turn of the cochlea of a fetal guinea-pig. The pregnant animal received subcutaneous injections of the solution of sodium salicylate for three weeks. Two of the cells show changes in the mitochondria similar to those in the cell of figure 4 *B*.

COMMENT

It is obvious that both quinine and salicylate act in such a manner as to alter the mitochondria in the various structures of the cochlear duct. The lower half of the cochlea is more prone to the action of the drugs than is the remainder. The administration of quinine to human beings in large doses produced a decrease in hearing acuity for low tones (Pohlman and Kranz [5]). One is hesitant to ascribe to animal experimentation the same course of events as occurs among human beings who suffer a loss of hearing following the taking of drugs. Undoubtedly, drug idiosyncrasies play a major rôle in the production of lesions within the cochlea. Nevertheless the emphasis on these cell changes which are found in animals seems justified.

5. Pohlman, A. G., and Kranz, F. W.: On the Effect of Certain Drugs, Notably Quinine, on the Acuity of Hearing, Proc. Soc. Exper. Biol. & Med. **20**:140, 1922.

The mitochondrial damage described for the structures of the cochlear duct is probably due to a direct action of the drug such as Wolff [6] has described for the effects of quinine on the retinal elements. He believes that quinine acts primarily as a general protoplasmic poison and in a second stage is responsible for spasm of the retinal vessels. The latter, he thinks, is due to an extreme deficiency in the supply of oxygen. Lurie [7] believes that drugs and certain bacterial toxins produce degenerative changes in the hair cells.

In spite of the similar findings for the two drugs there are certain discrepancies which are noteworthy. Quinine produces more profound lesions, as described for the stria vascularis and the hair cells of the organ of Corti. The spiral ganglion cells after treatment with sodium salicylate exhibit a greater degree of change than after administration of quinine.

Crowe, Guild and Polvogt [8] could find no pathologic lesions in 25 per cent of seventy-nine human ears which had revealed a gradual loss for high tones. They attributed the results either to cell changes impossible to demonstrate by the usual technical procedures or to lesions located within the complicated pathways of the central nervous system.

Confusion has arisen in the interpretation of the cochlear response, for which the ultimate explanation may be found in cell changes so fine as to be difficult of demonstration. Hallpike and Rawdon-Smith,[9] after severing the eighth nerve in the cat and allowing sufficient time for degeneration of the nerve, were able to demonstrate persistence of the organ of Corti, even though the cochlear response was absent. They were thus led to conclude that the response is nervous in origin. This view is contrary to that of Davis [10] and his co-workers, who regard the hair cells as essential. If an analysis of the constituents of the sensory or hair cells had been possible in these instances, an explanation might have been available. Unfortunately, with present technical methods all of the various types of cochleae do not lend themselves to such a study.

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6. Wolff, E.: The Causation of Quinine Blindness, Lancet **1**:1497, 1935.

7. Lurie, M. H.: Animal Experimentation on Hearing: Its Clues to the Prevention of Deafness, Tr. Am. Acad. Ophth. **40**:375, 1935.

8. Crowe, S. J.; Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High Tone Deafness, Bull. Johns Hopkins Hosp. **54**:315, 1934.

9. Hallpike, C. S., and Smith, A. F. R.: The "Wever and Bray Phenomenon": A Study of the Electrical Response in the Cochlea with Especial Reference to Its Origin, J. Physiol. **81**:395, 1934.

10. Davis, H.: Audition: III. The Physiological Phenomena of Audition, in Banister, H., and others: Handbook of General Experimental Psychology, edited by C. Murchison, Worcester, Mass., Clark University Press, 1934, p. 962.

SUMMARY AND CONCLUSIONS

The normal mitochondrial content of the cells of the cochlea and the changes produced after drug administrations have been studied in the cochleae of guinea-pigs.

Quinine bisulfate and sodium salicylate produce mitochondrial changes in the cells of the stria vascularis and in the sensory cells of the organ of Corti.

More drastic changes in the nature of chromatolysis and a loss of ground substance are evident in the spiral ganglion cells after injections of a solution of sodium salicylate than after injections of a solution of quinine bisulfate.

The alterations described usually are more pronounced in the basal turns of the cochlea, but over a longer period of time the apical turns may also become involved.

The cochleae of the fetal guinea-pig's ears exhibit similar but more profound changes than those of the adult animal.

ANOMALOUS VASCULAR LESION IN CEREBELLOPONTILE ANGLE

SEVERE NEURALGIC PAIN IN THE EAR AND PROFOUND NERVOUS DISTURBANCE; OPERATION AND RECOVERY

HAROLD I. LILLIE, M.D.

AND

WINCHELL McK. CRAIG, M.D.

ROCHESTER, MINN.

Pain in the ear from causes other than suppurative disease of the ear or its immediate appendages is frequently difficult to classify accurately. True neuralgic pain in the ear is infrequently encountered as a major complaint. Reichert [1] reported a case of tic douloureux of the ear, which he called tympanic plexus neuralgia. The patient, a young woman, had sharp, stabbing, excruciating pain deep in the external auditory canal, of such severity that she would cry out and cringe, holding her hand to her ear. The seventh, eighth and ninth nerves were exposed with the area under local anesthesia, and the pain could be reproduced only by stimulating the ninth nerve. For this reason the ninth, or glossopharyngeal, nerve was sectioned, and the patient was subsequently relieved of pain. We are reporting a similar case, in which a vascular lesion was present in the cerebellopontile angle.

REPORT OF CASE

A girl 18 years of age consulted one of us (Lillie) on Nov. 14, 1935, complaining of (1) severe neuralgic pain in the left ear, which occurred suddenly at frequent intervals and disappeared as suddenly as it occurred, (2) increasing deafness in the left ear and (3) a dull, continuous ache over the left side of the head.

The history revealed that these symptoms had begun suddenly three years previously without apparent cause, while the patient had been attending school. The pains had continued to recur intermittently throughout the day and night, and she was said to have had some fever. She remained in bed several days, and the fever subsided. The pain continued for two weeks and then suddenly stopped for a month, whereupon it recurred and gradually increased in frequency and severity. The patient noted nothing that could have caused the pain to recur other than

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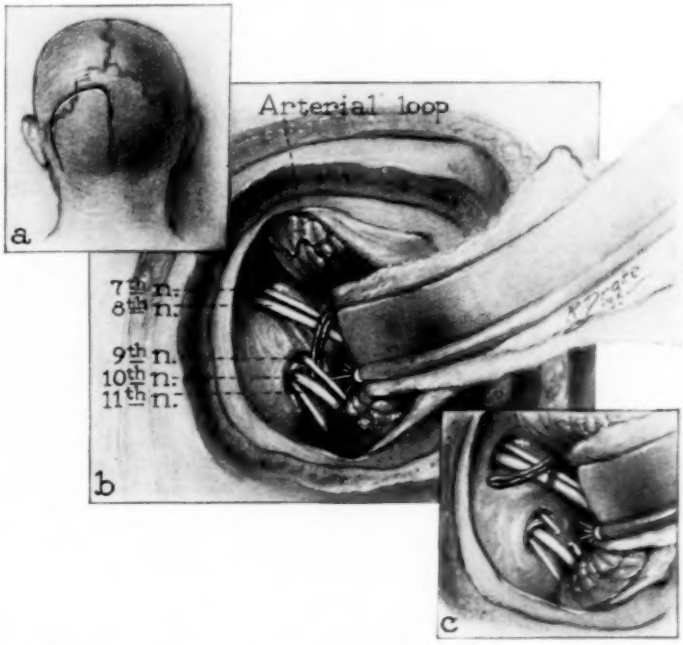
From the Section on Otolaryngology and Rhinology and the Section on Neurologic Surgery, The Mayo Clinic.

1. Reichert, F. L.: Tympanic Plexus Neuralgia: True Tic Douloureux of the Ear or So-Called Geniculate Ganglion Neuralgia: Cure Affected by Intracranial Section of the Glossopharyngeal Nerve, J. A. M. A. **100**:1744 (June 3) 1933.

manipulation of the ear. Therapeutic measures previously employed had given no relief. The patient had noted gradually increasing deafness for more than a year, the deafness having become profound; she had not noted vertigo or tinnitus.

The patient's facies and general demeanor made her appear to be of the stable type, and there was nothing to suggest that she was suffering from a neurosis. At intervals she would suddenly place her left hand to her ear; her face would assume the appearance of severe pain, and a moment or two later relaxation would take place. No objective pathologic conditions were encountered in the mouth, fauces, nasopharynx, nose or ears. There were no trigger zones in the pharynx or on the epiglottis. Cocainization of the pharynx and nasal ganglion did not control the pain. Insertion of a speculum in the ear, particularly when it touched



Drawings of the craniotomy, with *a* showing the incision; *b*, exposure of the cranial nerves and the position of the arterial loop, and *c*, division of the ninth nerve and elevation and displacement of the arterial loop, thereby relieving pressure on the eighth nerve.

the superior wall of the external auditory canal, caused the characteristic pain to recur. Functional examination of the ears revealed profound nerve deafness on the left and hyperactivity of both vestibular labyrinths. There was no perceptible anesthesia of the cornea. General physical and neurologic examinations gave negative results. Roentgenologic examination of the head with the patient in the Towne position revealed no evidence of a lesion at the left cerebellopontile angle. All laboratory tests gave negative results.

In arriving at a diagnosis, otalgia could be ruled out, as the teeth and nasopharynx were normal and cocainization of the nasal ganglion had not given relief,

nor was the pain suggestive of neuralgia of the vidian nerve. The progressive unilateral deafness of the nerve type suggested the possibility of a lesion in the cerebellopontile angle, although there was no corneal anesthesia, tinnitus, vertigo or loss of function of either labyrinth. Cases of tumors of the cerebellopontile angle have been observed in which one branch of the eighth nerve might be involved without affecting the other branch. It was suspected that the involvement in the present case might be similar to that in Reichert's case, because of the nature of the pain; however, the profound nerve deafness could not thus be accounted for. Because of the deafness and the persistence of pain, exploration of the left cerebellopontile angle was advised to rule out the possibility of an acoustic neuroma or some other pathologic lesion involving the eighth and ninth nerves.

Unilateral cerebellar craniotomy (fig. 1*a*) was carried out on November 21, with the patient under ether anesthesia and in the sitting position. The cerebellar lobe was easily elevated, and the left cerebellopontile angle was examined. There were no signs of an acoustic neuroma, and at first glance the seventh, eighth, ninth, tenth and eleventh nerves seemed to be normal in situation and in appearance. On closer examination, however, an anomalous artery could be seen looping over the eighth nerve and under the ninth nerve (fig. 1*b*). This was probably the anterior inferior cerebellar artery or one of its branches. The artery could not be elevated until the ninth nerve had been sectioned. After this was done, we were able to slip the arterial loop from its position between the eighth and the ninth nerve, thus relieving pressure on the eighth nerve (fig. 1*c*). The wound was closed in the usual manner, and as soon as the patient awakened from the anesthesia she stated that the pain was definitely gone; because it had been intermittent, however, we were not sure that relief was permanent until the following day.

For the first four postoperative days the patient was very dizzy, being unable to raise her head from the pillow; this condition gradually cleared, however, and she was allowed to get out of bed on the ninth postoperative day, at which time there were no signs of ataxia or nystagmus and thorough examination gave negative results except for anesthesia of the nasopharynx produced by sectioning of the ninth nerve. The patient was allowed to return home after the wound had healed. The pain was completely relieved, and her condition was excellent. At the time of her dismissal, no apparent change had taken place in the hearing in the left ear, but six weeks later she wrote that her left ear had returned to normal and that she could hear a watch tick and could use the telephone without pain and with apparently normal hearing.

SUMMARY AND COMMENT

Glossopharyngeal neuralgia is a clinical entity identified by a characteristic sharp, lancinating pain in the throat, which is projected into the ear and is produced by eating, drinking or irritation of the tonsillar region. The pain can be relieved by cocainization of the nasopharynx. Atypical glossopharyngeal neuralgia may be caused by a lesion of the cerebellopontile angle, as emphasized by Weisenburg[2] in his early

2. Weisenburg, T. H.: Cerebello-Pontile Angle Tumor Diagnosed for Six Years as Tic Douloureux: The Symptoms of Irritation of the Ninth and Twelfth Cranial Nerves, J. A. M. A. **54**:1600 (May 14) 1910.

description of the syndrome. In the case reported there were atypical features of glossopharyngeal neuralgia, which was not relieved by cocainization of the nasopharynx. Craniotomy was carried out because of an associated progressive deafness on the same side, and an anomalous vessel was found, which produced pressure on the eighth nerve and irritation of the ninth nerve. After dissection of this vessel and section of the ninth nerve pain was completely relieved, and in six weeks the hearing had apparently returned to normal.

VESTIBULAR TESTS

THEIR PRACTICAL APPLICATION

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PHILADELPHIA

The vestibular tests in themselves need no introduction, but a few introductory remarks about their application will not be amiss. There are many ways in which these tests may be utilized in various fields of medicine. It is my impression, however, that the tests are not utilized as frequently or with as much confidence as they deserve. The present situation is in marked contrast to the situation that existed when the tests were new and every one felt that miracles could be wrought with them. Presumably, disappointments following the overenthusiasm account for the swinging of the pendulum in the opposite direction.

It occurs to me that there are other reasons to account for the limited and infrequent use of these tests. Otologists, who are mostly concerned with them, seem to feel that the tests are too complicated to be bothered with and that one can probably do without them. This feeling is enhanced by the apparent lack of confidence manifested by some neurologists and neurosurgeons in the results of these tests. At one time, however, confidence existed to a marked degree.

To the otologist one can say that the complexity of the vestibular tests has been exaggerated. They are no more complex than tuning fork tests, which no otologist would exclude from his armamentarium. Furthermore, the vestibular tests are just as useful and at times even more important than the tuning fork tests. It is true that some of the experimental and physiologic explanations of these tests are at times confusing. But it is necessary to memorize only a few simple tests and the normal reactions together with the significance of a few abnormal reactions to be prepared to make use of the tests. I dare say that the average otologist employs tuning forks as a routine measure to good advantage in his practical work but probably knows little of the physics, construction, variation and decrement of these forks. This, of course, does not interfere with the clinical application of the tuning fork tests.

Read before the Section of Otolaryngology of the College of Physicians of Philadelphia, March 20, 1935.

From the Department of Neuro-Otology, the Graduate School of Medicine, the University of Pennsylvania, service of Dr. Coates.

There is also a wrong impression among otologists that the vestibular tests are of value only when an intracranial lesion is present. Strange as this may seem, many neurologists and neurosurgeons have obtained a similarly wrong impression that the tests are of no value at all in neurology. It seems to me, however, that otologists can utilize the tests in many ways in their field without bothering to apply them in making a neurologic diagnosis. I shall try to point out some of these applications.

As to the neurologists, one recalls the enthusiasm that they showed for these tests a few years ago, but as time went on diagnoses by these tests were frequently in error and biased with neurologic findings, so that many neurologists lost confidence in them. As a result the useful and correct diagnoses obtained by the tests were lost sight of. Now and then one meets a neurologist who smiles knowingly at what he calls the uselessness of the vestibular tests.

There is one point, however, that should be taken into consideration by the neurologist: Certain things happen when a patient who has an intracranial lesion is given the vestibular tests which do not happen when a normal person is tested. This merits a neurologic explanation. Much significance is attached to the finding of a paretic ocular muscle as a diagnostic aid; but when the same muscle "goes wild" in a wrong direction when it is caused to move in a vestibular test, the result is often not taken seriously.

One reads records of neurologic examinations in which absence of the Hoffmann sign or of the Oppenheim sign is always reported. Hoffmann's sign is flexion of the thumb when one of the fingernails is pinched; Oppenheim's sign is flexion of the toes when the median surface of the leg is stroked. These are supposed to be signs of a lesion of the pyramidal tract. Their presence or absence is always recorded with great reverence; yet recently I read a report of ten obscure cases of tumor of the brain in which various diagnostic procedures were described but no mention was made of an attempt to obtain information by means of the vestibular tests. Frequently the manifestations elicited by the vestibular tests are so far from normal, for instance when one ocular muscle pulls up instead of laterally or one group of muscles acts instead of another or the muscles of the two eyes do not work in unison or fail to work at all, that one wonders that these phenomena do not intrigue the diagnostic acumen of the neurologist. However, I sympathize with the neurologist, for the reason that occasionally the neuro-otologist in his ignorance of neurologic conditions attempts to foist a diagnosis on the neurologist which jars the neurologic mind. I think, however, that if the neurologist and the neuro-otologist conferred, they could be of assistance to each other. Without the neurologist's cases and

without his advice neuro-otology cannot progress. But to abdicate the entire subject of neuro-otology to the neurologist would be impracticable, as otologic problems are frequently deeply concerned.

Be that as it may, from the otologist's point of view the neurologist need not accept the tests at all, as there are many uses to which the otologist can put them that will be of help.

APPLICATION

In attempting to apply the tests it is well to memorize the accepted standards of normal responses and interpret the results on that basis, bearing in mind the normal variations. The normal responses are few in number, and if one is as familiar with them as with the multiplication table the findings can easily be interpreted. Naturally it is always better to understand the subject well if unusual problems are to be interpreted, particularly in view of the fact that the present standards are not altogether final. Otologists who do not care to understand the subject in its entirety can still make use of the findings by means of a working knowledge of the normal as well as some of the abnormal responses.

In the application of these tests in otology one need take into consideration only (*a*) the diminution of any or all of the normal responses, or, hypo-activity, (*b*) the total absence of responses from one or both sides and (*c*) the hyperirritability of the labyrinth, as manifested by hyperactivity. An acquaintance with the anatomic structures of the inner ear is, of course, assumed, whether an otologist is interested in the vestibular tests or not. Abnormal types of responses are chiefly of value in the diagnosis of intracranial lesions.

The manifestation of hypo-activity is useful chiefly as confirmation of a similarly depressed function of the cochlea. When there is an impairment of hearing of the nerve type, the corresponding impairment of the vestibular function serves as objective confirmation of the nerve character of the deafness. This is desirable when malingering is suspected and is useful in medicolegal procedures involving litigation, in which objective findings are relied on more than subjective ones. With regard to medicolegal procedures, it may be well to mention that claims for damages in cases of accident or of employer's liability are often made on the basis of deafness and vertigo. These symptoms frequently follow injuries to the head. When objective signs of depressed function, as manifested by the vestibular findings, are demonstrable credence is more likely to be given to the claimant who may be perfectly honest and yet is suspected of malingering. There are even some abnormal types of findings occasionally obtained in a person who has sustained an injury to the skull and whose only complaint is that of vertigo, no neurologic signs being present.

Certain diseases and drugs that have a predilection for the eighth nerve, such as syphilis, diabetes and meningitis and quinine, may have their effects objectively demonstrated by a depressed vestibular function.

Occasionally, when one bears in mind the arterial blood supply of the labyrinth, the complaints of vertigo and deafness in a patient may be traced to the circulatory nature of his disturbance if the proper combination of depression is found. Thus, the end-artery of the labyrinth which supplies the part of the cochlea in which high-pitched sounds are perceived supplies also the vertical canals. When there is impairment of the vertical canals together with a loss of hearing of high-pitched notes in the proper type of person, the cause of dizziness may possibly be suspected as being arteriosclerotic. The symptoms grouped as Ménière's syndrome also may in this way be shown to be manifestations of circulatory disturbance in some instances.

Absence of vestibular responses on one side is a valuable finding from the otologist's point of view. Vestibular tests are utilized in patients who have had signs of labyrinthitis to determine whether the labyrinthitis is purulent or serous. With the return of some of the vestibular function, one is not likely to believe the disease to have been suppurative and would be deterred from operating on the labyrinth. The return of the cochlear function is not so reliable, as it is subjective and the patient's statement that he hears may be the result of hearing with the opposite ear.

Before radical operation on the mastoid is undertaken, knowledge of the condition of the labyrinth is of utmost importance. The demonstration of an erosion into the labyrinth during operation when the labyrinth is active calls for careful avoidance of further encroachment, but if the labyrinth shows a total loss of function and of irritability labyrinthectomy may be indicated.

I recall one case of simple mastoidectomy in which previous knowledge of the state of the labyrinth might have been of value. Several operations had been performed on the mastoid for recurring infections. The structures were badly distorted, and when a canal was opened during an operation its character and direction could not be definitely determined. It may have been a semicircular canal or the facial canal. About ten or twelve hours after the operation facial paralysis developed. No trauma to the nerve could have been inflicted as the paralysis came on late. The labyrinth could not have been opened, as no vestibular disturbance appeared after operation. The vestibular tests a short time after the operation indicated that the labyrinth was functionless and devoid of irritability. Then the question arose as to whether the labyrinth was in that condition before the operation (and labyrinthectomy indicated) or whether the labyrinth was ablated during the operation. As the peration was simple mastoidectomy, no vestibular tests

had been performed in advance. As to prognosis, one was in a quandary. If the facial canal had been opened, the possibility of permanent damage was to be considered. If another canal had been opened, the paralysis would probably disappear, as it developed some time after operation. If the canal had been a fistula into the labyrinth (the function of the labyrinth being previously impaired owing to prolonged surrounding infection), its destruction was probably completed. Perhaps the labyrinth was functionless and nonirritable before operation. Vestibular tests in advance would have supplied the necessary information.

So, one need not find interest in the vestibular tests merely for the diagnosis of intracranial involvement. There are enough otologic problems involved to warrant attention to them.

A functionless, nonirritable labyrinth in an unconscious person suspected of having an otitic abscess of the brain may indicate that the abscess is in the cerebellum rather than in the cerebrum. The labyrinth is a frequent route of origin of a cerebellar abscess. This is another instance in which vestibular tests would be of value in solving an otologic problem.

Hyperactivity of the labyrinth may be of diagnostic importance chiefly in the determination of the cause of vertigo. There are instances in which severe vertigo, staggering and falling arouse suspicion of the presence of intracranial disease. Normal but hyperactive responses suggest that the vertigo is probably of toxic origin and that an extraneous cause should be sought. The appearance of vertigo in a patient with a chronic infection of the ear when the vestibular phenomena are hyperactive may indicate the presence of perilabyrinthitis and the necessity for radical mastoidectomy.

THE DIAGNOSIS OF INTRACRANIAL LESIONS

The diagnosis of a condition thought to be due to an intracranial lesion should be approached with the full understanding that most of the information concerning such lesions is empirical and based on signs which have previously but not always been proved to indicate the presence of certain intracranial lesions. No histopathologic data have been recorded showing the appearance of the brain stem when a tumor of the brain is present and when the patient manifested some atypical form of nystagmus. It is possible for a lesion of the brain to be present without the manifestation of signs of vestibular involvement. It is also possible to find signs of vestibular involvement pointing to an intracranial lesion but no other neurologic signs to indicate the presence of such a condition. One would hardly blame a neurosurgeon for not operating on the basis of these signs alone. The cases in which positive signs are present should always be regarded with suspicion, and further developments awaited. In some cases the only signs of an intracranial

lesion have been those of vestibular involvement, but months later other neurologic data have been obtained at operation and a lesion has been found in the location indicated by the signs referable to the vestibule. On the other hand, records sometimes show that a patient has manifested signs of vestibular involvement which pointed to the presence of an intracranial lesion but that the symptoms disappeared and no operative treatment was instituted. An interesting fact is that all patients who show signs of vestibular involvement suggesting an intracranial lesion complain principally of headache or vertigo. It is possible that temporary intracranial conditions may produce these signs. All in all, caution is required in making a diagnosis.

Before attempting to determine the site of a lesion normal conditions should be recalled in detail. When the responses are normal, slightly diminished or exaggerated, nothing can be added to the picture, even though neurologic signs are definite. There are phases in the vestibular responses which have a negative value. For instance, when the vestibular responses are good one would hardly expect to find a lesion in the cerebellopontile angle. It is not likely that a lesion of the posterior fossa exists if the patient is very sensitive to the tests. Such problems do arise in neurologic diagnosis, and the tests can be helpful even though they show no positive results.

The important positive findings which suggest the presence of an intracranial lesion are (*a*) perverted nystagmus, (*b*) inverted nystagmus, (*c*) vertical nystagmus, (*d*) oblique nystagmus, (*e*) dissociated ocular movements and (*f*) conjugate deviation. Some less certain but nevertheless suggestive signs are extremely large amplitudes of nystagmus, a fibrillary type of ocular movements, poor pelvic girdle reactions, marked disproportion between vertigo and nystagmus, absence of vertigo when nystagmus is present and, vice versa, absence of responses from the canals in one plane when responses are obtained in another plane.

As a general rule, the presence of some signs indicative of an intracranial lesion and diminution of the responses of nystagmus, vertigo, past pointing and sensitivity speak for an infratentorial lesion. When hyperactivity is noted, a supratentorial lesion is more likely to be present. When diminution or exaggeration is more or less equal on the two sides, the lesion, whether supratentorial or infratentorial, is most likely in the midline. When one side is affected, it is generally true that the supratentorial lesion is located on the opposite side of the abnormal findings, but an infratentorial lesion is probably located on the same side. When there is a perversion of the responses from the horizontal canal the lesion usually proves to be on the same side.

There are other data which may or may not be of value but which should be considered. For instance, conjugate deviation often suggests

a lesion on the side toward which the eyes are drawn. Then there are groups of combinations of findings suggestive of the location of the lesion. The most reliable of these is the syndrome indicative of a lesion of the cerebellopontile angle, e. g., a total lack of responses from the side involved, including deafness, absence of sensitivity to the tests, absence of response from the vertical canals of the opposite side or, just as frequently, a perverted response of some sort, either a perverted or an inverted nystagmus, from either the vertical or the horizontal canals.

COMMENT

I believe that there is enough value in the vestibular tests to merit their serious consideration by the otologist and the neurologist. The neurologist and the neuropathologist can be of the greatest help in making an effort to explain the bizarre manifestations on the same basis as other neurologic phenomena are explained—by neuropathologic study.

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THRESHOLD OF FEELING IN THE EAR IN RELATION TO SOUND PRESSURES

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The threshold of feeling in the ear resulting from stimulation by relatively great sound pressures has been investigated principally by physicists interested in acoustic phenomena in relation to hearing. Previous study of this threshold has been concerned chiefly with persons with normal hearing. This paper extends these investigations into the field of pathology.

A review of the literature on the threshold of feeling reveals the following conclusions: *A*. The threshold of feeling probably bears no relation to stimulation of the auditory nerve.[1] *B*. Approximately the same values of sound pressures are required to elicit the threshold in both normal and abnormal ears.[1] *C*. The pressure required to elicit the threshold of feeling in the ear is about the same as that required to excite the tactile nerves in the finger-tips.[1] *D*. The quality of the sensation varies with the frequency and the pressure.[1] *E*. The intersecting points of the threshold of audibility and the threshold of feeling determine both the upper and the lower frequency limit of audibility in human ears.[1] *F*. It is difficult to distinguish between the sensation of hearing and that of feeling where the thresholds of audibility and of feeling intersect.[1] *G*. "At the lower limit of audibility the excursions of the diaphragm and ossicles of the middle ear are probably so large that the nerves feeding these movable parts are stimulated and therefore the measurements at very low frequencies probably give data related to the pathological condition of the apparatus of the middle ear and this independently of the stimulation of the auditory nerve."[1] *H*. If the intensity of a tone is increased sufficiently, a level of intensity is reached at which the ear responds with a sensation of tickle.[2] *I*. The

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1. Wegel, R. L.: The Physical Examination of Hearing and Binaural Aids for the Deaf, Proc. Nat. Acad. Sc. **8**:155, 1922.

2. Fletcher, H.: Physical Measurements on Audition and Their Bearing on the Theory of Hearing, Bell Syst. Tech. J. **2**:289, 1923; J. Franklin Inst. **196**: 289 (Sept.) 1923.

threshold of feeling is as definite or as critical as the threshold of audibility.[2] *J.* This threshold serves as a practical upper limit for the intensities of tones which can be perceived or tolerated by the ear, since pain results from increasing sound pressures above the values necessary to reach the threshold of feeling.[2] *K.* Deaf persons without perception of sound experience the threshold of feeling.[3] *L.* It is presumed that the sensation is tactual, due to activation of the nerves of the tympanic membrane.[4] *M.* Sound pressures of sufficient magnitude to elicit the threshold of feeling occasionally stimulate the receptors of the static sense.[5] *N.* The exact end-organs responsible for the sensation are unknown.[5]

APPARATUS AND TECHNIC

The experimental set-up consisted essentially of a General Radio type 613-A beat frequency oscillator, a high quality audio frequency amplifier and a modified Western Electric no. 555 receiver. The acoustic output of the receiver was calibrated by means of the artificial ear[6] developed at the Bell Telephone Laboratories, Inc., in terms of decibels above 1 bar of sound pressure when 1 milliampere of current flowed through the voice coil of the receiver. In the present study a thermocouple milliammeter was connected in series with the voice coil to indicate the magnitude of the current flowing through it.

Inspection by means of a General Electric type HC-10-B1 cathode ray oscillograph of the wave form of the test frequencies at the plates of the push-pull output tubes of the amplifier with maximum permissible currents through the voice coil of the receiver revealed no observable departure of the receiver input from the sinusoidal wave form.

A history and physical examination of the ears and a complete functional examination of the hearing, including the making of audiograms by means of a Western Electric 2-A audiometer, were first obtained for all patients and observers. An ear was considered normal if it had a normal history, a normal-appearing tympanic membrane, a positive reaction to the Rinne test with the 256 double vibration fork and a loss not exceeding 10 sensation units, or decibels, for any of the eight frequencies generated by the 2-A audiometer.

The receiver was suspended so that its position was easily adjustable, and each observer was instructed to press the cap of the receiver against his ear as tightly as possible without causing discomfort. He was told that the loudness of each tone in the receiver would be increased gradually until a new sensation in addition to that of sound would be experienced. Each observer was requested to indicate as soon as any extra-auditory sensation was perceived and to describe it to the best of his ability. Three reports were taken for each test frequency, and the values were averaged.

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3. Jones, I. H., and Knudsen, V. O.: Facts of Audition, Ann. Otol., Rhin. & Laryng. **34**:1013, 1925.

4. Banister, H.: Hearing: I. The Foundation of Experimental Psychology, edited by C. Murchison, Worcester, Mass., Clark University Press, 1929, p. 907.

5. Wegel, R. L.: Physical Data and Physiology of Excitation of the Auditory Nerve, Ann. Otol., Rhin. & Laryng. **41**:740, 1932.

6. Inglis, A. H.; Gray, C. H. G., and Jenkins, R. T.: A Voice and Ear for Telephone Measurements, Bell Syst. Tech. J. **11**:293, 1932.

OBSERVATIONS

Absolute Values of Sound Pressures Required to Stimulate the Threshold of Feeling of the Normal Ear at Different Frequency Levels.—Curve *A* of chart 1 shows the average values of sound pressures in terms of decibels above 1 bar necessary to produce the sensations which defined the threshold of feeling in thirty-eight normal ears of adults at the frequencies indicated. As only six of the nineteen observers reported extra-auditory sensations at the maximum permissible output of sound pressure of the receiver at 4,096 cycles, 72.4 decibels above 1 bar, the value for this frequency is not shown. Curve *B* shows similar data for forty-eight ears in the original study of the threshold of feeling.[1] Curve *C* shows the results obtained from a study of eight (?) ears,[5] and curve *D* shows data for an unstated number of ears.[7]

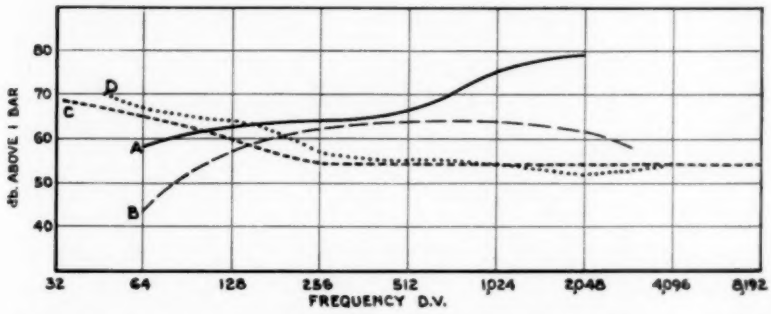

Chart 1.—The thresholds of feeling in decibels above 1 bar: *A*, Lierle and Reger, 1935 (thirty-eight ears); *B*, Wegel, 1922 (forty-eight ears); *C*, Wegel, Riesz and Blackman, 1932 (eight [?] ears), and *D*, Riesz, 1933.

The discrepancies of the data shown in chart 1 are not readily explained. Contributing variable factors were the methods of calibration of the receivers or sound reproducers used, the number of observers, the instructions to observers, the manner of presentation of the stimuli and the closeness with which the receiver was held against the ear, especially at the lower levels of frequency.

In the present study the intensity was increased gradually by means of a continuously variable attenuation control until the observer reported the perception of an extra-auditory sensation. The effect on the threshold in question of increasing the intensity in discrete steps or intervals of 1 or more decibels at a time was not studied.

Individual Variations in the Normal Threshold of Feeling.—One of the earlier investigators [2] reported that "experiments show that the inten-

7. Riesz, R. R.: The Relationship Between Loudness and the Minimum Perceptible Increment of Intensity, J. Acoust. Soc. America **4**:211, 1933.

sity for this sensation is approximately the same for various individuals and the results can be duplicated as accurately as those for the minimum intensity value."

The accompanying table shows the magnitude of the individual differences of the threshold of feeling for the thirty-eight ears with normal hearing. The numbers in the vertical columns which are marked with

Individual Differences in the Threshold of Feeling in the Thirty-Eight Normal Ears

Decibels Above 1 Bar	Frequency, Cycles per Second					
	64	128	256	512	1,024	2,048
86	..	..	..	..	..	1
85	..	..	..	..	..	2
84	..	..	..	..	..	1
83	..	..	..	..	5	2
82	..	..	..	..	1	5
81	..	..	..	..	3	3
80	..	..	..	..	5	7
79	..	..	..	..	2	3*
78	..	..	..	..	1	2
77	..	..	..	..	5	5
76	..	..	..	..	1	2
75	..	1	1	..	1*	..
74	..	..	..	2	2	..
73	..	1	2	1	..	3
72	..	1	..	4	1	..
71	..	..	5	1	2	..
70	..	..	..	2	..	1
69	2	5	..	1	1	..
68	..	..	3	3	1	..
67	1	2	1	8	1	..
66	..	..	5	2*	..	..
65	2	4	1	1	1	..
64	..	2	2*	2	..	..
63	4	1*	3	2	2	..
62	..	3	6	..	..	..
61	2	7	..	1	1	..
60	2	4	3	3	..	..
59	4	..	1	..	..	..
58	3*	3	..	3	..	1
57	8	..	..	..	..	..
55	4	..	..	..	1	..
53	1	..	4	..	..	..
52	..	4	..	1	..	..
49	..	..	..	..	1	..
47	4	..	..	..	..	..
46	..	..	1	1	..	..
41	1	..	..	..	..	..

* This figure denotes the average values for the threshold of feeling in terms of decibels above 1 bar for the thirty-eight ears at the indicated frequency levels.

asterisks indicate the average values of sound pressure required to stimulate the threshold at the different frequency levels. Reference to the table shows that at a frequency of 64 cycles extra-auditory sensations were experienced in only three of the thirty-eight ears at the average value of sound pressure necessary to elicit the threshold at this frequency, 58 decibels above 1 bar. Also, at 64 cycles the minimum pressure required to produce extra-auditory sensation was 41 decibels, and the maximum was 69 decibels, above 1 bar. In general, variations between the ears of the same subject were much less than variations from subject to subject.

Aside from possible error in the calibration of the receiver, variations in the closeness with which the receiver was held against the ear probably contributed the major source of error in the study. It was observed that if the silent receiver was placed against the auricle in a certain position and with a certain pressure, a sensation resulted similar

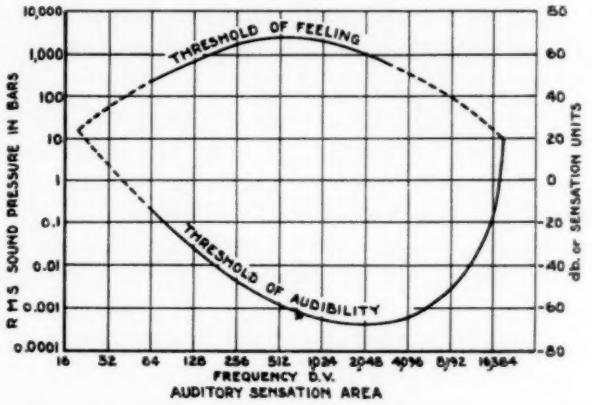

Chart 2.—Auditory sensation area. R. M. S. indicates root mean square.

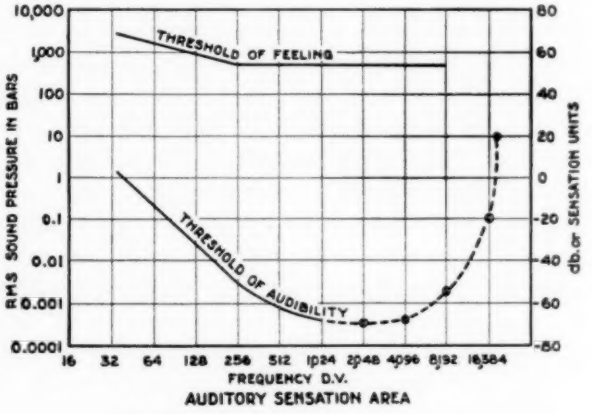

Chart 3.—Auditory sensation area.

to that due to stopping the ear by pushing the tragus against the opening of the external auditory canal, so that the external canal and the air space external to the diaphragm of the receiver seemed to form a closed system. It was observed that under this condition the values of sound pressure necessary to stimulate the threshold of feeling were slightly less, especially for the lower frequencies, than when the receiver was pressed in a haphazard fashion against the ear. However, because of differences in the size and shape of auricles and the difficulty of

ascertaining whether a closed system existed while measurements were being obtained, an attempt to record data only while there was a closed system was abandoned.

After consideration of the previously mentioned sources of error, it is believed that the magnitude of the individual differences may have been due to some extent also to variations in the distribution and supply of the sensory nerve endings in the tympanic membrane. This statement is based on the fact that three subjects were encountered during the course of the study in whose ears no extra-auditory sensations were evoked by the maximum permissible sound pressures from the receiver. One of these subjects had normal-appearing tympanic membranes and normal hearing; one had slightly retracted tympanic membranes and an approximate 50 per cent bilateral loss of hearing, and the third had scarred and retracted membranes, with an approximate 40 per cent bilateral loss of hearing, as calculated from the audiogram made with the 2-A audiometer. It was observed that the membranes of these six ears lacked the usual sensory equipment associated with these structures. When they were touched and slight pressure was applied to them with a cotton applicator, only faintly unpleasant sensations were elicited. The reaction of these six membranes suggested that marked variations probably occurred in the distribution and supply of the sensory nerve endings to the tympanic membrane.

Comparisons of the Threshold of Feeling in Normal Ears and in Ears with Complete Loss of Hearing.—To determine the relationship of stimulation of the threshold of feeling to stimulation of the auditory sense, eight persons were studied who had normal-appearing tympanic membranes but complete bilateral loss of hearing. Five of these subjects had lost their hearing as a result of meningitis, and three were congenitally deaf. The following tabulation shows the average values of sound pressure necessary to elicit extra-auditory sensations at the indicated frequency levels in the sixteen ears:

Frequency, cycles	64	128	256	512	1,024	2,048
Decibels above 1 bar....	60.6	66.4	68.3	71.5	76.8	81.0

Extra-auditory sensation was experienced in only seven of the sixteen deaf ears at a frequency of 4,096 cycles, with the maximum permissible sound pressure at this frequency level, 72.4 decibels above 1 bar.

Comparisons with curve *A* of chart 1 or with the average normal values of sound pressure for the threshold of feeling given in the table (marked with an asterisk) indicate that the average values for the deaf ears closely approximate the equivalent values for the normal ears. Individual differences or variations in the minimum and maximum val-

ues of sound pressure required to stimuate the threshold of feeling also are of approximately the same order of magnitude as in the normal ears.

It is interesting to note that several persons encountered in the study who were presumed to be completely deaf as the result of infection or congenital defect were found to possess auditory perception at the lower frequency levels with sound pressures less than those necessary to produce extra-auditory sensations. Some of these subjects, who reported and described the lower tones as hums or buzzes, experienced only extra-auditory sensations at the higher frequency levels. The data obtained from such subjects were not included in the tabulations in this paper.

Since the values of sound pressure required to reach the threshold of feeling are approximately the same for normal ears and for ears with complete loss of hearing but with intact tympanic membranes, it seems that the sensations which define the threshold of feeling do not result from stimulation of the eighth nerve.

Location and Identity of the Receptors Responsible for the Threshold of Feeling.—It was observed in patients with one intact tympanic membrane but with a perforation in the other that the minimal sound pressures which elicited extra-auditory sensations in the ear with the intact membrane did not stimulate the threshold of feeling in the ear with the perforated membrane. The maximum permissible output of sound pressure of the receiver was not sufficient to produce extra-auditory sensations in seven of fifteen ears with perforated tympanic membranes. Hence no data for sound pressure can be given for the ears with perforated membranes. (The maximum permissible outputs of sound pressure of the receiver were approximately 10 decibels greater than the average normal values for the threshold of feeling for the four lowest test frequencies and 7 decibels greater for the two highest test frequencies.) Visual inspection or correlation of the size of the perforation with the values of sound pressure for the threshold of feeling, when experienced, led to the general observation that the larger the size of the perforation the greater the sound pressure required to stimulate the threshold, especially at the higher frequency levels. In general, it was impossible with the maximum sound pressure available to reach the threshold of feeling in ears in which the size of the perforation was as great or greater than approximately one-half the area of the membrane itself.

Three ears with the tympanic membrane so markedly retracted that it appeared to be in apposition to the medial wall of the cavity of the middle ear also were studied. It was impossible to elicit extra-auditory sensations in these ears with the maximum permissible outputs of sound pressure of the receiver. Also, it was impossible with the maximum

ranges of sound pressure and frequency to produce extra-auditory sensations in sixteen of twenty-one ears which had completely healed after a radical mastoidectomy.

These results strongly suggest that the sensations which define the threshold of feeling in normal ears are due to the stimulation of receptors within the tympanic membrane. It was observed that the values of sound pressure for the threshold of feeling of persons with normal hearing and those of deaf persons with normal-appearing tympanic membranes were practically identical. The threshold was raised for ears with perforated membranes and was not stimulated with the maximum sound pressures available in sixteen of twenty-one ears after a radical mastoidectomy. One of the most conclusive pieces of evidence was the discovery that the three persons with intact membranes who did not experience the threshold of feeling apparently lacked normal sensitivity of the membrane, as determined with the cotton applicator.

Microscopic anatomic and clinical observations further strengthen the conclusion that the threshold of feeling is due to the stimulation of receptors within the tympanic membrane. It is well known that the normal tympanic membrane is liberally supplied with free nerve endings, generally believed to be pain receptors. Wilson [8] also described a second type of receptor within the tympanic membrane, which he classified as a modified pacinian corpuscle. Extra-auditory sensations arising from stimulation by sound pressure in an ear lacking the tympanic membrane probably are due to the stimulation of contact receptors within the epithelium which lines the combined cavity of the middle ear and the external auditory canal.

Nature of the Extra-Auditory Sensations Which Define the Threshold of Feeling.—The character of the extra-auditory sensations which identify the threshold of feeling varies according to the frequency of the sound pressure as well as from observer to observer at the same frequency level. The most common descriptive term employed for a sound pressure below 1,024 cycles was "tickle." A few observers reported that the lower frequencies elicited a sensation in the ear similar to that experienced when a vibratory massage is given about the head. Other observers reported the following sensations as the result of stimulation by the lower frequencies: itch, sting, wind blowing in the ear, tingling or numbness similar to the sensation resulting when the foot "goes to sleep," flutter, pinpricking, a drilling against something back in the ear, walls of the canal vibrating, going under water, probing back in the ear, pinching back in the ear and something poking against the drum. At frequency levels above 1,024 cycles the most usual descriptive terms were pressure and pain. At these higher frequency levels some

8. Wilson, J. G.: The Nerves and Nerve Endings of the Membrana Tympani of Man, Am. J. Anat. **11**:101, 1911.

observers described the sensations as a sharp ache, a burning back in the ear, a pushing inside the ear, water in the ear, wind blowing into the ear, diving into deep water, stinging pain, pinprick in the ear, pushing with a cotton probe against the ear drum and the sensations experienced when a fast-moving elevator stops and starts.

Sensations of vertigo were not common to all observers but were reported frequently for sound pressures at high frequency levels. In a few instances the vertigo lasted for a few hours and was sufficiently intense to cause nausea. After stimulation some observers reported various types of tinnitus, lasting for several hours in a few instances. No permanent injurious effects were reported. A few observers noticed a light tickle or sensation of contact at the point where the ear-piece or cap of the receiver touched the ear. The frequency intensity levels at which this sensation was experienced on the auricle were not recorded as data relative to the true threshold of feeling.

Once the threshold of feeling was elicited, an increase of sound pressure a few decibels above this value quickly produced exceedingly painful sensations, no matter what the frequency level. When extra-auditory sensations were experienced by an observer lacking the tympanic membrane, the threshold of feeling usually was not so sharply defined, localized or critical; the sensations experienced were rarely painful and were more diffused throughout the canal.

Although the sound pressures required to elicit the threshold of feeling in the ear are approximately equal to those required to produce tactile sensations at the finger-tips under certain conditions, the qualities of the sensations at these two diverse anatomic points are not similar. A pressure which produces a tickle in the ear may be felt at the finger-tips as a very light "touch" or sensation of contact or as a current or jet of air blowing against the finger. Pressures which cause intense pain in the ear do not elicit even mildly unpleasant sensations at the finger-tips. Sound pressures from a source several feet from the ear but sufficiently great to stimulate the threshold of feeling do not produce tactile sensations at the finger-tips.

Relation of the Threshold of Feeling to Amplification of Sound for Hard-of-Hearing Ears.—Fletcher [9] stated that persons with a loss of hearing between 80 and 110 decibels require powerful aids to hearing involving amplification by vacuum tubes; they cannot understand speech at all without such equipment. Persons with loss of hearing greater than 110 decibels cannot use such aids. "The vowel sounds hurt them before the consonant sounds are above their threshold." This is due to the fact that vowel sounds produce much greater sound pressures than consonants. It is believed that the foregoing statement may be modified

9. Fletcher, H.: Can We Scientifically Advise Patients as to the Effectiveness of Hearing Aids? Ann. Otol., Rhin. & Laryng. **41**:727, 1932.

in relation to the person whose threshold of feeling is raised above normal because of reduced sensitivity of the tympanic membrane, perforated membrane or absence of the membrane.

During the course of the study we encountered two exceedingly hard-of-hearing persons who lacked membranes and who preferred to listen to music amplified from phonograph records in head phones at an intensity level which stimulated the threshold of feeling in normal ears. Consequently it is believed that an exceedingly hard-of-hearing person possessing perception of sound over the frequency range necessary to permit intelligibility of speech sounds but with a raised threshold of feeling can tolerate and use a higher level of amplification of speech than a person with an equivalent hearing loss but a normal threshold of feeling.

Relation of the Threshold of Feeling to the Functional Examination of Hearing.—The concept of the threshold of feeling as developed in this paper does not complicate or modify in any way the conventional functional examination of the auditory acuity of persons with sufficient hearing for ordinary conversational purposes unless attempts are being made to determine the absolute upper and lower frequency limits of audibility. The fact is axiomatic that the upper and lower frequency limits of audibility are functions of the values of the sound pressure of the respective stimulus frequencies used. It is well known that greater sound pressures are necessary to reach the threshold of audibility near the lower and higher frequency limits than to reach this threshold over the middle range of audible frequencies.

Due consideration, however, must be given the concept of the threshold of feeling when testing the auditory acuity of persons who are exceedingly hard of hearing. To measure all degrees of loss of hearing it is necessary to employ sound pressures within the limits of the audible frequency range from the level of the threshold of audibility to that of the threshold of feeling. The tuning forks commonly used by the otologist are rarely capable of producing sound pressures of sufficient magnitude to stimulate the threshold of feeling, with the possible exception of large forks generating frequencies below 256 cycles. It is possible, however, to produce sound pressures sufficiently great to stimulate this threshold over a much wider frequency range by electrical methods utilizing reproducers of sound driven by alternating electric currents. The examiner must be certain that at high intensity levels the sensation experienced by the patient is that of perception of sound and is not extra-auditory. If there is the possibility of a doubt as to the quality of the sensation experienced by the patient, he should be required to describe what he is reporting. This procedure of course is most reliable for intelligent adults.

MacFarlan [10] reported that "when a child is examined it is always uncertain whether the vibration of the testing instrument is heard or felt. . . . It is not possible to discriminate between tactile and auditory impressions received from the audiometer when at full strength. When the 3A audiometer is used, tactile interference appears at a setting of 85 to 90 per cent loss in hearing. When the receiver is in contact with the auricle, the vibration is felt at even weaker intensities." In a later article MacFarlan [11] so clearly pointed out the methods of distinguishing between perception of auditory and of extra-auditory sensation that this phase of the threshold of feeling in relation to the functional examination of hearing needs no further elaboration. It should be noted, however, that MacFarlan used the phrase "tactile sense" with a more inclusive connotation than we use the term threshold of feeling. The phrases "threshold of feeling" and "extra-auditory sensations" have not been used to include the sensations of vibration arising from mechanical contact at the point where the receiver meets the auricle or the sensations of air currents perceived about the auricle owing to mass displacement of air molecules.

Comments on the Method Used for the Determination of the Percentage of Hearing.—The phrase "percentage of hearing" is being used more and more often in quantitative statements relative to the degree of impairment of the hearing function. Knudsen and Jones [12] stated: "Otologists, as well as patients, are more accustomed to think in terms of 'percentage hearing,' rather than in decibels of hearing loss."

If sound pressures must be increased to the extent that the threshold of feeling is stimulated before the perception of sound occurs (before the threshold of audibility is reached), a 100 per cent loss of hearing is said to exist. This is due to the fact that pressures greater than those necessary to produce feeling are decidedly uncomfortable and limit the usable maximum intensity of sound that can be tolerated by the ear. Quantitative values for the threshold of feeling therefore are necessary factors in the determination of the percentage of hearing. At present, calculations of percentage hearing or percentage loss of hearing usually are based on the original data obtained by Wegel,[1] shown as curve *B* in chart 1. Curve *C* shows comparable data by Wegel, Riesz and Blackman [5] in a more recent study. Calculations based on the data in curve *B* show that a hearing loss of 30 decibels for 64 cycles is equivalent to 50 per cent loss of hearing for this fre-

10. McFarlan, D.: Distinguishing Between Tactile Sense and Audition in the Deaf Child, Arch. Otolaryng. **5**:507 (June) 1927.

11. MacFarlan, D.: Tactile Sensations as Related to Hearing Testing and Hearing Impressions Through Nerves Other Than the Eighth, Ann. Otol., Rhin. & Laryng. **42**:680, 1933.

12. Knudsen, V. O., and Jones, I. H.: Basic Principles Underlying Tests of Hearing, Laryngoscope **45**:1, 1935.

quency. According to the data of curve *C*, however, the loss is only 37 per cent.[13] This difference emphasizes the necessity for further study and subsequent standardization of the values on which calculations of the percentage of hearing are based, especially the data on the threshold of feeling.

Inspection of chart 1 shows that at a frequency of 64 cycles the individual differences in the sound pressures required to reach the threshold of feeling in normal ears varied from 47 to 69 decibels above 1 bar. In the ear in which the threshold of feeling was 47 decibels above 1 bar at 64 cycles a hearing loss of 30 decibels is equivalent to a 50 per cent loss. But in the ear in which the threshold of feeling was 69 decibels above 1 bar at the same frequency level a hearing loss of 30 decibels is an approximate loss of only 37 per cent.

If one adheres to the definition of complete hearing loss as previously stated, it is apparent that in order to make the most accurate statement possible regarding the percentage of hearing of a given person for a certain frequency, it is necessary to know the intensity value at which his threshold of feeling is stimulated. This is particularly true in cases in which the threshold of feeling is markedly raised above the normal values owing to pathologic change, perforation or absence of the tympanic membranes.

To avoid confusion in statements regarding percentage of hearing or percentage loss of hearing in scientific papers, it seems advisable to state at the same time the values for the threshold of feeling in decibels above the threshold of audibility or zero level on which the calculations for the percentage are based.

SUMMARY

The present paper corroborates the previous work on the threshold of feeling in normal ears. The additional experimentation with pathologic ears definitely establishes the following points:

1. The sensations which identify the threshold of feeling arise from receptors within the tympanic membrane and epithelium lining the external auditory canal and middle ear.

2. A person who lacks the tympanic membranes can tolerate higher levels of speech amplification without discomfort than can persons with normal thresholds of feeling.

3. Additional work is necessary to establish with greater accuracy the average normal threshold of feeling.

4. Statements relative to percentage of hearing or percentage loss of hearing should be accompanied with the values of the threshold of feeling on which the calculations are based.

13. A sound pressure of 1 bar at 64 cycles is approximately 13 decibels above the average normal threshold of audibility at this frequency level.

BACTERIOLOGIC STUDIES IN ACUTE AND IN CHRONIC MAXILLARY SINUSITIS

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Our reasons for presenting the results of our bacteriologic studies of material obtained from the maxillary sinus are as follows: 1. The studies have been continued over a period of twelve years; this time factor has given us opportunity to compare the results of repeated bacteriologic tests in many cases and also to compare the bacteriologic and clinical findings. 2. The results of the studies stress the importance of assuming the presence of definite pathologic changes in antrums the irrigation returns of which are characteristically clear and of albuminous-like consistency. 3. The observations demonstrate the value of a direct microscopic examination of the exudate for comparison with the growth obtained in culture.

We have done all of our own laboratory work; the services of a technician have not been used for any part of the studies. The facilities of the National Institute of Health, Washington, D. C., were used for the early investigations. Our early results on the clear, albuminous-like exudates were reported by one of us (A.) before the Osler Society of Washington, D. C., in 1926.

A good review of the literature on this subject has been published by Balmer,[1] and unnecessary repetition will be avoided. The literature is somewhat disappointing, however, in that the data presented are not sufficiently definite. Long lists are recorded of bacterial parasites and saprophytes isolated from the maxillary sinus. The organisms most frequently encountered are mentioned, but rarely does one find notations as to the particular organism occurring alone, or in preponderance. Rarely also does one find any mention of microscopic studies, either cytologic or bacteriologic, of the exudates removed. The organisms chiefly reported are, in their order of frequency: Staphylococcus albus, Streptococcus pyogenes (haemolyticus), Streptococcus viridans, Diplococcus pneumoniae (Pneumococcus), diphtheroids and Encapsulatus pneumoniae (Friedländer's bacillus).

1. Balmer, Frederick B.: The Relation of Clinical to Bacteriologic Observations in Normal and in Diseased Maxillary Antrums, Arch. Otolaryng. **14**:440 (Oct.) 1931.

The study has been divided into two series, A and B. Series A represents the results obtained from the clear, albuminous-like returns, and series B, those obtained from the definitely purulent types.

METHODS

Method 1.—The nasal chambers were thoroughly irrigated with physiologic solution of sodium chloride, after which films and, in most cases, cultures were made from the middle meatus and from the inferior meatus. Sterile cotton swabs (wrapped on fine copper wire) saturated with a 10 per cent solution of cocaine in physiologic solution of sodium chloride were placed under the inferior turbinate, and after anesthesia was established a second smear and culture were made from the site of puncture and a sterile Coakley trocar was introduced into the antrum. A sterile glass syringe was then attached to the adapter and a small amount of physiologic solution of sodium chloride placed in the sinus with the patient's head inclined toward the shoulder of the same side. This fluid was slowly withdrawn; films were prepared and cultures made at once. Freshly prepared blood agar plates (p_H 7.4) and a semisolid 0.1 per cent dextrose medium [2] were found most useful for the cultural studies. The latter medium serves well for partial anaerobiosis.

Method 2.—All our recent studies have been made by the following method: The preliminary steps were similar to those described under method 1, with the exception that cocaine hydrochloride flakes saturated in a 3 per cent solution of ephedrine sulfate were used to produce anesthesia. A small curved Coakley trocar and cannula, the handle of which was wrapped with sterile gauze extending up over the opening of the adapter, were introduced beneath the inferior turbinate, and the trocar was withdrawn with its sterile wrapping elevated over the nose and downward over the mouth. Into the cannula was then inserted a sterile Yankauer eustachian tube applicator wrapped with a few fibers of cotton on either the ringed or the straight end. Sterile microscopic slides were then smeared and cultures made from the same swab. If more material was desired additional applicators were introduced into the antrum.

MICROSCOPIC STUDIES

A good microscopic examination of properly prepared and stained films is indispensable. Thick films are best, since in many cases the organisms are not abundant and they are usually enmeshed in the thicker masses of exudate. Loeffler's alkaline methylene blue, phenol fuchsin and the modified Gram stain were used. In such films we were able to see not only the preponderant organism but also the types of cells present, phagocytosis, etc. A comparison of films prepared from the material obtained from the middle meatus and from the area of antral puncture with those of material obtained inside the antrum is of great value in solving the problem as to the etiologic significance of any particular organism.

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2. Enlows, Ella M. A.: A Sugar-Free Medium for Fermentation Studies, Pub. Health Rep. **38**:2129 (Sept. 14) 1923; also reprint no. 868.

Series A.—At the time we began our studies there was a great deal of controversy concerning the nonpurulent type of exudate. Only a few observers believed that there were pathologic changes present. In fact, at the present time there still are otolaryngologists who insist on the demonstration of frank pus as an indication of infection in the sinuses. Pus is a term applied to the exudate from infected or irritated tissues, and it may vary all the way from firm, cheeselike masses to an almost water-like thinness. The otologist who has studied microscopically exudates from the middle ear knows this to be true. Following myringotomy often only a thin, water-like fluid escapes, but when this is examined microscopically it is seen to be teeming with bacteria and to contain a fairly large number of polymorphonuclear leukocytes. Many factors are, no doubt, involved in these different infectious processes. Some of them may be dependent on the type of organism. It is known that Str. pyogenes (haemolyticus) when the incitant of infection in the mucous membrane usually causes a thin, hemorrhagic or serous exudate in the early stages of the infection and that later this exudate frequently becomes frankly purulent. On the other hand, Str. viridans in such situations seems to provoke a much slighter outpouring of leukocytes but a greater loss of tissue fluids.

Our clinical findings and the results of various forms of treatment forced the conclusion that infection must be present in the patients from whose antrums these clear, albuminous-like irrigations were obtained. They presented symptoms in many instances identical with those of patients from whom purulent exudates could be demonstrated: supra-orbital and temporal pain, tenderness of the entire maxillary bone on the affected side, often extending into the teeth, which in some cases were needlessly sacrificed; general malaise, dizziness, gastrointestinal disturbances, etc. Two patients in this series were suffering from retrobulbar neuritis, which cleared up completely when the antrums received the appropriate treatment after the demonstration of definite bacterial infection.

Observations: Thirty-four cases were studied in this series; in all, the condition was chronic. Str. viridans was found to be the only organism present in twenty-four, or 72 per cent, of the cases. Of the remaining ten cases, no growth was obtained in four, although the films showed streptococci of the viridans type were present. In six of the ten cases the infection was of mixed type; Str. viridans was the predominant organism, but Staph. albus, of the hemolytic variety, was also present.

The microscopic films prepared from the antral exudate in this series were very interesting. Large numbers of polymorphonuclear leukocytes embedded in a gelatinous matrix were observed. The num-

ber of bacteria seen varied from only an occasional pair or short chain in one or two fields of the entire slide to enormous numbers, usually occurring in groups. The morphology of the streptococci found in such exudate is quite similar to that which is seen when these organisms are grown under ideal cultural conditions. They are usually of good size, and typically lanceolate pairs are seen alone or in short chains. There is never any capsule, and when films are made later from the same patient, when improvement is noted, many pleomorphic forms may be seen. When improvement has set in the exudate may also become somewhat more purulent. These pleomorphic forms strongly resemble the true diphtheroids and may be readily mistaken for them. Str. viridans shows a strong tendency toward this pleomorphism when grown under certain cultural conditions. One such strain (S 25) isolated during this study from a chronically infected maxillary sinus was carried for several years as a striking example of the rod form. Single cell studies were made and the lanceolate and rod forms produced at will according to the nutrient medium used.

Series B.—The frankly purulent material from the first eighty-nine patients was obtained by method 1; material from the remaining forty-three was obtained by method 2. The results are summarized in the table.

Summary of Results for Series B

	Positive Results		
	Cases of Acute Infection	Cases of Chronic Infection	Total
Str. anhaemolyticus * with Staph. albus (haemolyticus)	..	1	1
Str. viridans, pure culture	30	18	48
Str. viridans, with Staph. albus (haemolyticus)	11	11	22
Str. viridans, with Str. pyogenes	5	4	9
Str. viridans with D. pneumoniae	4	0	4
Str. pyogenes (haemolyticus), pure culture	10	4	14
Str. pyogenes (haemolyticus), with Staph. albus	1	0	1
Str. pyogenes (haemolyticus), with D. pneumoniae	1	0	1
D. pneumoniae, pure culture	14	9	23
D. pneumoniae, with Staph. albus	0	2	2
Staph. albus (haemolyticus), pure culture	0	1	1
Staph. albus (haemolyticus), with Str. viridans	0	1	1
Encapsulatus pneumoniae with Str. viridans, and Staph. albus	0	2	2
Bor. Vincenti with Staph. albus	0	1	1
Bor. Vincenti with Staph. albus and Str.	..	..	..
Bor. Vincenti with Staph. viridans	0	1	1
Bor. Vincenti with Staph. albus and D. pneumoniae	0	1	1
	76	56	132

* The streptococci have been classified according to their reactions on blood agar mediums, the fermentation of inulin and solubility in bile. In a few cases serologic tests were made. Fermentation of carbohydrate does not give results accordant with agglutination and absorption of agglutinin reactions. Only ten of the strains of pneumococci were typed. Sixty per cent were of type II; the remainder were of type IV. Borrelia (Spironema) Vincenti was identified by staining reactions of the direct microscopic film. In each slide examined from these three patients there were both spirals and fusiform bacteria.

COMMENT

The results obtained from any bacteriologic study can prove misleading if adequate control is not maintained. If the exudate obtained from a sinus is allowed to stand at room temperature for several hours or is placed in an icebox for from ten to fourteen hours, as is often the case, inaccuracy results. This was determined by actual experiment. A number of species of the staphylococci multiply rapidly at room temperature, thus obscuring the organism originally predominating. The low temperature of the icebox completely prevented growth of Str. viridans in seven cases and allowed growth of only two colonies on one of three blood agar plates in one other case. Examination of the microscopic film indicates clearly what organism predominates, and that particular organism is obtained in culture if proper precautions are taken. When such cultures are made late in the course of an acute infection in the presence of marked phagocytosis, the growth is often scanty or absent.

The absence of Micrococcus catarrhalis did not surprise us since it is not found frequently in any region of the nose. Some years ago in an effort to obtain a pure culture of this organism for class demonstration, cultures and smears were made from fifty-seven nasal cavities. Carefully selected and adjusted culture mediums were employed, and material from healthy and diseased mucous membranes was included. The results were entirely negative.

Much effort has been expended by investigators in an effort to rule out the bacteria present on the lateral nasal wall at the site of puncture. Balmer [1] has described an adequate technic devised for this purpose. There is no doubt as to the trustworthiness of his procedure, but there are a fair number of patients whose mucous membranes do not react well to the preparations employed (solution of epinephrine and of metaphen and metaphen ointment). In a nose already congested and edematous it is well to avoid adding further insult. In view of the results we have obtained with the methods described, we believe that our technic is sufficiently elaborate for all routine purposes. We have repeated our cultural and microscopic studies of material from one patient at weekly intervals as many as five times and have obtained similar results each time. The safeguard of accuracy lies in the initial smear and culture from the site of puncture. And this should be made a second time just before the trocar is introduced; this involves no additional disagreeable procedure for the patient. The only added labor is the taking of the preliminary cultures and the precaution of sterilizing a wrapped Coakley trocar and a few Yankauer applicators.

In all the cases studied the preponderant organism in the films and culture obtained from the middle and inferior meatuses was the same

as that in the material from the antrum. The films and cultures made from the site of puncture gave negative results in about 10 per cent of the cases; in other cases only one or two colonies of staphylococci, usually Staph. albus (hemolytic variety), grew in the cultures made from this area. In all cases the film and culture from beneath the middle turbinate were identical with those obtained from the maxillary sinus.

SUMMARY

Bacteriologic and microscopic studies were made of the exudates obtained from the maxillary sinuses of one hundred and sixty-six patients. Thirty-four of the cultures were of the clear, albuminous-like type; the remainder were definitely purulent. Str. viridans was isolated in pure culture from seventy-two, or 43 per cent, of the total number of exudates examined. D. pneumoniae was found in pure culture in twenty-three, or 14 per cent, of the exudates. Str. pyogenes (haemolyticus) ranks third; it occurred alone in fourteen, or 9 per cent, of the exudates. Staph. albus (hemolytic type) was found in pure culture only once. No obligate anaerobes were found in either series.

RELATION OF THE STAPEDIUS REFLEX TO HEARING SENSATION IN MAN

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AND

H. B. PERLMAN, M.D.

CHICAGO

The contractions of the stapedius muscle in man in response to sound stimuli were first observed by Luescher,[1] in 1929, with the aid of the aural microscope. Since that time only a small number of cases in which this reflex has been observed have been reported in the literature.

It has appeared, therefore, necessary to extend these observations and to collect a larger series of reports of cases in which the reflex was visible in order to determine more definitely the relationship between the reflex and the hearing sensation.

Our observations in the clinic during the past several months have shown that a visible stapedius reflex is not a rare occurrence and that it may frequently be demonstrated during a routine examination.

In a number of patients with a perforation in the upper posterior quadrant of the drum, the stapes or stapedius tendon is visible. If the tendon is observed through a Siegle speculum and acoustic irritation (e. g., Bárány noise box) is introduced into the opposite ear, the movements frequently can be seen.

By means of this simple method, twenty-five cases have been observed in which we have been able to make determinations of the threshold for the hearing sensation and for the visible stapedius reflex in response to sound. In many cases the thresholds for both air and bone conduction have been determined.

It is convenient to divide the cases into groups for the purpose of illustration and to select certain cases to represent each group.

OBSERVATIONS AND REPORTS OF CASES

Group 1.—In this group there was good hearing in one ear, with a stapedius reflex in the opposite ear which could easily be visualized. Several cases of this type have been studied. Three cases are reported here.

From the Division of Otolaryngology of the Department of Surgery of the University of Chicago.

This work was done in part under a grant from the Douglas Smith Foundation for Medical Research of the University of Chicago.

1. Luescher: Ztschr. f. Hals-, Nasen- u. Ohrenh. **23**:105, 1929.

Case 1.—Miss E. W., 19 years old, was admitted to the clinic because of nasal disturbances. There was no history so far as the ear was concerned. Examination revealed a normal ear drum on the left side and a small, dry perforation in the posterior superior quadrant of the right membrana tympani. Provided the head was kept in a certain position, the stapedius tendon could be seen through the perforation. With the telephone membrane of the audiometer (Western Electric 2-A) at the patient's left ear, the curve for hearing was recorded (chart 1, solid line). Immediately after the curve was plotted, a Siegle speculum was inserted into the right ear, and the stapedius tendon was observed. The Siegle speculum was used only because of the lens. The telephone membrane remained at the left ear. The intensities were now determined which produced the first visible movement of the tendon. As shown on the chart (broken line), the first movements could be seen if the tone was about 65 decibels stronger than the threshold of hearing.

Chart 2 shows the same observations for bone conduction. The bone-conducted sounds were produced in the audiometer. The applicator of the audiometer was

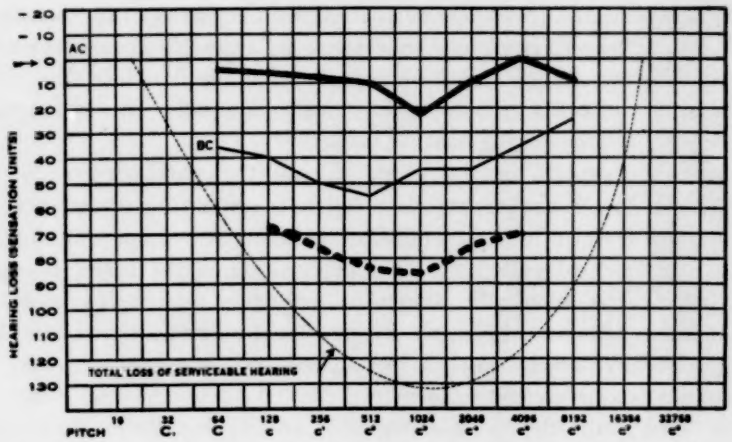

Chart 1 (case 1).—The upper line represents the curve for hearing of the left ear by air conduction, tested with the 2-A audiometer. The dotted line indicates the threshold of contraction of the right stapedius tendon, with the telephone membrane of the audiometer on the left ear.

attached to the microphone of a mechanism to aid hearing by bone conduction, and the oscillator was kept on the mastoid process. Although the tone is amplified in the instrument for bone conduction to an unknown degree, a fact which deprived us of the use of our decibel unit, we consider the application of the instrument as serviceable for our purpose. With the oscillator on the upper teeth, both the threshold of hearing and the threshold of the stapedius reflex for bone conducted sounds were determined.

Case 2.—Miss E. L., aged 28, complained that the left ear had been discharging for ten years. The membrana tympani was mostly destroyed, with the stapedius tendon visible. Chart 3 shows the curve for the threshold of hearing of the right ear, and the threshold of response of the left stapedius reflex when the audiometer receiver was at the right ear. The intensities produced by the audiometer were sufficient to excite a visible reflex for only three frequencies.

CASE 3.—Mr. M. R., 29 years old, complained that his right ear began to discharge three years previously. Examination revealed a posterosuperior marginal perforation, with a cholesteatoma. The left ear was normal. The curve

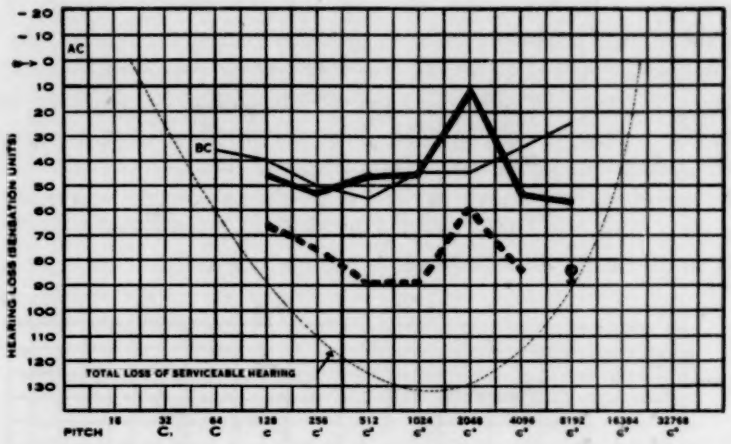

Chart 2 (case 1).—The curve for hearing by bone conduction. The oscillator of the instrument for aiding bone conduction was on the teeth of the upper jaw. The heavy line indicates the hearing sensation, and the lower line, the curve for the reflex.

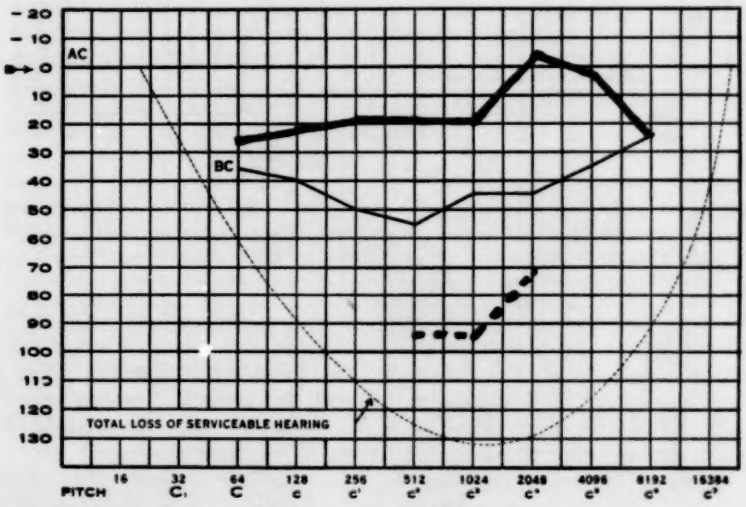

Chart 3 (case 2).—The upper line indicates the threshold for hearing in the right ear. The broken line shows the threshold for the left stapedius reflex when the audiometer receiver was at the right ear.

for hearing of the left ear is represented by the unbroken line in chart 4. The right stapedius tendon started visible movements if the sound was increased to 80 or 85 decibels. When this chart is compared with chart 1, the distance between

the two curves is seen to be greater. As the chart shows, the intensities produced by the audiometer for 64, 128 and 256 decibels were not strong enough to excite a visible reflex response in the right stapedius tendon.

Group 2.—In this group of cases the intensities produced by the audiometer, the telephone receiver being used, either did not produce a visible reflex or caused a reflex at only one or two frequencies. This occurred in cases of bilateral impaired hearing or in cases in which either the motility or the visibility of the reflex had been handicapped by cholesteatoma, adhesions or granulations.

In carrying out the tests in this group, the audiometer was connected with an amplifier and loud speaker in order to produce sufficient intensities to obtain the threshold of the visible reflex.

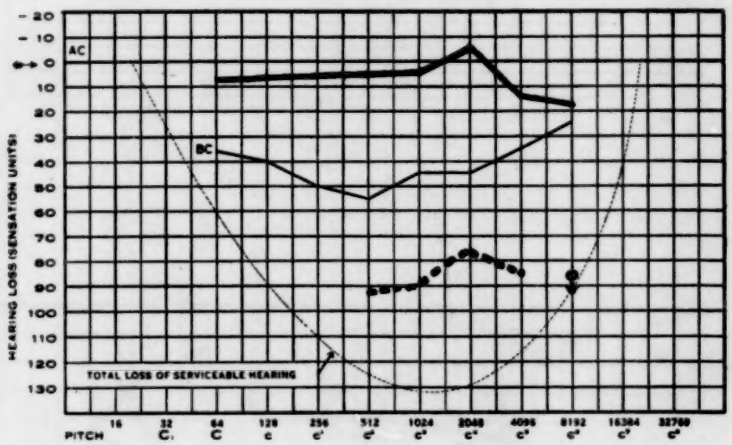

Chart 4 (case 3).—The upper line indicates the hearing sensations when the telephone membrane of the audiometer was kept on the left ear. The broken line represents the threshold of contraction of the right stapedius tendon under the same conditions.

CASE 4.—Mr. W. W., aged 25 years, showed a large, dry defect of the right membrana tympani, with a visible stapes. Because of the negative result with the telephone membrane of the audiometer, the tones were amplified. The loud speaker was placed 1 foot (30 cm.) behind the patient. Chart 5 shows the threshold of hearing as measured with the audiometer and the threshold of the visible stapedius reflex when amplified tones of the same instrument were used. The increased intensities necessary to make the reflex visible were therefore greater than the distance between the curves indicates. It is significant that the form of the curve for the reflex coincides closely with the form of the curves for hearing.

Group 3.—Another group of patients was observed in whom hearing by air conduction had been greatly impaired by bilateral lesions

of the middle ear and in whom the reflex for air-conducted sounds was unsatisfactory, even after amplification. The patients in this group still showed good bone conduction, and the reflex contractions could

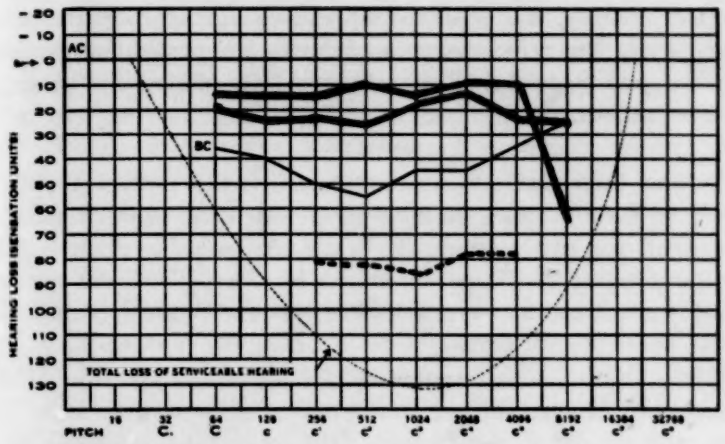

Chart 5 (case 4).—The two heavy lines represent the hearing sensation of both ears, the upper line being that for the right ear. The dotted line indicates the right stapedius reflex produced by tones amplified and delivered by a loud speaker.

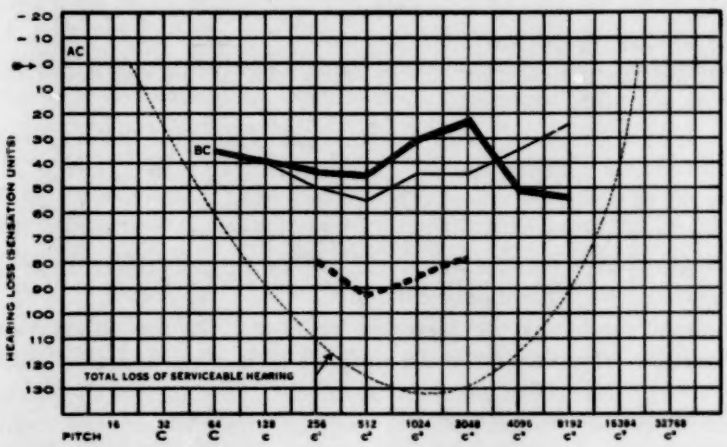

Chart 6 (case 5).—The heavy line represents the hearing sensation and the dotted line the stapedius reflex for bone-conducted sound in a patient who did not show the reflex by air conduction. The applicator of the instrument for aiding bone conduction was placed on the left mastoid area. The left stapedius tendon was observed.

be demonstrated in response to bone-conducted sound. Chart 6, made by observations on such a patient, shows the threshold of hearing by bone conduction and also the threshold of the visible stapedius reflex.

Effect of General Anesthesia.—An interesting observation may be mentioned here which may be interpreted as a test of the reflex nature of the movements of the stapedius tendon. One patient, in whom movements of the stapes were easily visible in response to sounds of moderate intensity, was observed while under general anesthesia with nitrous oxide. The reflex disappeared during this period but reappeared when the patient awoke. This corresponds to the results of experiments on rabbits.

COMMENT

We have been unable to see the stapedius reflex through the normal drum,[1a] or even in cases of atrophy in which the drum is unusually transparent, for two reasons: 1. While the ossicular chain is intact, the movement of the stapes produced by the pull of the tendon is extremely small and may not be visible with the Siegle speculum (contraction under normal circumstances is practically isometric).

2. The absorption and scattering of light by the ear drum, even in the most favorable conditions, is so great as to prevent accurate vision of the tendon and its movements. This cannot be overcome by the use of magnification.

Successful attempts to overcome the dispersion of light by the ear drum and so to render the reflex movements of the stapedius tendon visible through the intact ear drum have been reported by one of us (H. K.[2]) by the use of a solution of glycerin, water and potassium iodide applied to the surface.

All the patients observed in this series had varying degrees of pathologic change in the middle ear.

In such a group of cases certain factors must be taken into consideration which affect the reflex response: (1) the hearing acuity of the ear to which the sound stimulus is applied, both for air-conducted and for bone-conducted sounds and (2) the nature and degree of the pathologic change present which affects either the motility of the parts or the visibility of the movements.

Certain conditions are therefore necessary for the observation of the acoustic stapedius reflex in man. The hearing function must be satisfactory. The reflex arc must be intact. The stapes or the stapedius tendon must be visible, and the motility must not be prevented by adhesions or any other pathologic condition.

As regards the local pathologic changes in the middle ear, two types of cases have been distinguished: 1. Those in which there is a per-

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1a. Since this paper was submitted for publication we have observed one patient in whom the stapedius reflex in response to sound could be seen through the normal tympanic membrane.

2. Kobrak: Ztschr. f. Hals-, Nasen- u. Ohrenh. **32**:8, 1932.

foration, without destruction of the ossicles. In these cases the incudostapedial joint was intact. Either movement of the body of the tendon or movement of points of light on the tendon could be observed. 2. Cases in which the incus was absent or separated from the stapes. In these cases the pull of the stapedius tendon produces a visible tilting movement of the stapes. Analogous conditions prevail in the rabbit.

As all the patients had some degree of pathologic change in the middle ear, affecting the visibility of the reflex to various degrees, the thresholds of the reflex were observed to vary in relation to the hearing sensation in individual cases and in all instances were higher than would be obtained under optimum conditions for observing the reflex.

The significant finding in all the cases which have been studied has been the close parallelism between the hearing sensation and the reflex.

From the observations which we have been able to make, it appears safe to state that the stapedius reflex is an integral part of the human ear, comparable in function to the light reflex of the pupils.

Experiments on animals have revealed the anatomy of the reflex (Pollak,[3] Hammerschlag[4]).

The afferent branch is the acoustic nerve; the central part is represented in the acoustic nuclei and in association fibers with the trigeminal and facial nerves. The efferent branch for the tensor reflex is the trigeminal nerve, and for the stapedius reflex, the facial nerve. The reflex arch works both crossed and uncrossed; that is, stimulation of one side is followed by simultaneous contraction of both sides, as with the pupillary light reflex.

The importance of the acoustic stapedius reflex for practical clinical purposes is, at present at least, very limited. Observation of the reflex can be used, however, to determine the presence of hearing in persons suspected of malingering or in children. The chief value of the reflex at present is that it can be used as an indicator of hearing in connection with otologic investigations in animals.

In former papers we have reported experiments on animals in which the tensor and stapedius acoustic reflexes were used as indicators of hearing. The advantages of this fairly simple objective method for the determination of hearing for both air-conducted and bone-conducted sound have been previously pointed out. The important bridge between the reflex in experiments on animals and the hearing sensation is furnished by patients who can be tested regarding both the hearing function and the reflex activity.

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3. Pollak: Med. Jahrb. **1**:555, 1886.
4. Hammerschlag: Arch. f. Ohrenh. **47**:251, 1899.

SUMMARY

A series of twenty-five cases are described in which the acoustic stapedius reflex has been observed. Former observations on the stapedius reflex in man have been corroborated and extended.

The reflex contractions of the stapedius muscle in response to acoustic stimuli are a normal physiologic phenomenon acting primarily as a protective mechanism and bearing a definite relation to the hearing sensation. The acoustic stapedius reflex in man is abolished with general anesthesia.

A COMPARISON OF INTRADERMAL AND INTRAMUCOSAL TESTS IN CASES OF ALLERGIC RHINITIS

LLOYD D. LINTON, B.S.
ST. LOUIS

My purpose in this paper is to compare the results secured by intradermal tests with those secured by intramucosal tests.

MATERIAL AND METHOD OF STUDY

Sixty-five cases in which the condition was diagnosed as allergic rhinitis were studied. The patients were not selected. In half of the cases there were complications, such as asthma, headache, suppuration of the nasal sinuses or deflected septums. The cutaneous tests were made by Miss Zelma Alvis. Every effort was made to get accurate and reliable results. The technic for the intramucosal tests was the same as that previously reported,[1] except that the use of a 1 per cent solution of cocaine in the nose was discontinued; further, more than one set of tests per day were made provided the allergen used in the first test did not react within forty-five minutes.

Approximately 95 per cent of the positive reactions to the intramucosal tests begin within thirty minutes after the administration of the allergen.

The patients were first studied by Dr. Dean or his associates. Histories were carefully taken, and examinations were meticulously made. Cutaneous tests and cytologic studies of the blood and nasal discharge preceded the intramucosal tests and the bacteriologic study. An effort was made to confirm the positive cutaneous reactions by intramucosal tests. The mucous membrane was tested with all allergens with which the patient came in contact throughout the attack of allergic rhinitis. In seasonal allergic rhinitis the seasonal allergens were the ones used.

Twenty-nine of the sixty-five patients said there was a history of allergy in the family; fifteen said that there was not, and twenty-one were more or less uncertain as to the existence of allergy in the family.

OBSERVATIONS

Only one patient with definitely positive cutaneous reactions gave negative intramucosal reactions. Eighteen of the sixty-five patients were nonreactive to cutaneous tests; ten of these had no cutaneous reactions, and eight of them had from one to four plus-minus reactions.

From the Department of Otolaryngology, Washington University School of Medicine. This work was made possible by the Ball Research Fund.

1. Dean, L. W.; Linton, L. D., and Linton, C. S.: An Intramucosal Test for Hypersensitivity in Allergic Rhinitis, Ann. Otol., Rhin. & Laryng. **34**:317 (June) 1935.

Each patient gave one or more positive intramucosal reactions. In only five cases did the intradermal and the intramucosal reactions show 100 per cent agreement.[2]

The two following cases are typical:

W. B. suffered from allergic rhinitis and gave nine plus-minus cutaneous reactions. Two of the nine allergens used for the cutaneous tests produced positive intramucosal reactions. Feathers did not cause any cutaneous reaction but produced clinical symptoms with the intramucosal tests, and the avoidance of feathers gave the patient complete relief.

R. L. also had allergic rhinitis. He gave the following reactions to the intradermal tests: Ragweed, 3 plus; poplar, 3 plus; willow, 2 plus; goldenrod, 1 plus; oak, 1 plus, and sweet green pepper, 1 plus. Pepper and house dust produced plus-minus reactions. Poplar, goldenrod, sweet green pepper and house dust failed to produce positive intramuscosal reactions.

Of the sixty-five cases of allergic rhinitis studied, in thirty-three there were no complications, and in five the condition was complicated by headache in addition to the allergic symptoms, in two by headache and nasal polyps, in three by headache and asthma, in three by asthma and nasal polyps, in twelve by asthma and in seven by nasal polyps.

The intramucosal tests caused the following reactions, which accompanied the nasal symptoms: Thirteen patients suffered from headache, usually one side; in these thirteen patients thirty-five headaches occurred after intramucosal tests. Seven patients complained of a sensation of stoppage of the ears or of itching in the ears. The symptoms in the ear accompanied twenty-two tests. Five patients tested with different allergens had six asthmatic attacks. In three patients pain in the eye developed nine times. Three patients had mild pharyngitis; two had coughing spells six times; one complained twice of rheumatism-like pains and five were nauseated.

In eight of the thirteen cases in which headaches were produced, one of the complaints on admission was headache. Eight inhalants and ten foods were the offending allergens producing the thirty-five headaches. Six of the seven patients who had reactions in the ear had a history of trouble with the ears. Seven foods and six inhalants caused the reactions in the ears. All five of the patients in whom asthmatic attacks developed after the intramucosal tests gave a history of asthma.

The sixty-five patients gave a total of 240 positive cutaneous reactions and 290 positive intramucosal reactions. Only 111 of the positive cutaneous reactions, or 46.25 per cent, were confirmed by the intramucosal reactions.

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2. Rinkel, H. J., and Balyeat, R. M.: The Localization and Specificity of Cellular Sensitization, J. Allergy **3**:567 (Sept.) 1932.

The following tabulation shows the allergens in the order of frequency of positive reactions:

Inhalants

Intramucosal Tests	Intradermal Tests
Ragweed and feathers	House dust
House dust	Ragweed
Orris root	Tree pollens
Grasses	Feathers and grasses
Wool and tobacco	Horse dander
Tree pollen and animal hair	Animal hair
Horse dander and kapok	Wool
	Orris root, tobacco, kapok

Foods

Intramucosal Tests	Intradermal Tests
Whole wheat	Milk
Milk	Chocolate
String beans	Egg and wheat
Tomatoes, oranges	Oranges, cottonseed
Eggs	Rye
Beef, oats	Oats, black pepper, chicken
Fish, chocolate, pork	Sweet green pepper
Chicken, carrots, lettuce, cornmeal, coffee	Mixed nuts, rice, bananas
Spinach, asparagus, cottonseed, black pepper, mixed nuts, apples	Turnips, lamb, spinach
Potatoes, rice, cabbage, lamb, peas, bananas	Tomatoes, cornmeal, lettuce, peas, peaches, plums, carrots, malt, grapefruit, fish and potatoes
Peaches, turnips, rye, plums, celery, etc.	Cucumber, coffee, apples, asparagus, potatoes

SUMMARY

The reactions to intradermal tests are not a reliable index of the sensitivity of the nasal mucous membrane.

Seven patients had symptoms in the ears on entering the clinic; in six of them the symptoms were induced by the intramucosal injection of allergen in the nose.

Asthmatic attacks were induced by intramucosal tests in some asthmatic patients.

Headaches were produced in eight of the ten cases in which headache was an entrance complaint.

No one allergen was predominant in producing positive reactions when the symptoms produced were other than those of uncomplicated rhinitis.

The offending allergens can be determined in most persons with negative cutaneous reactions by intramucosal tests.

HEMANGIOMA OF THE NASAL SEPTUM

REPORT OF THREE CASES

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CLEVELAND

Granuloma of the nasal septum affords an interesting problem for differential diagnosis as well as for treatment. The diagnosis is of the utmost importance for avoiding faulty treatment. All too often inadequate or improper therapy aggravates the lesion, if it does not actually incite a fatal outcome.

The most common forms of granuloma arising from the nasal septum are hemangioma, papilloma, carcinoma, fibroma, polypi, tuberculoma and rhinoscleroma. Any granuloma of the nasal septum not arising from infection or from trauma by a foreign substance is casually considered as malignant in origin because it bleeds freely when touched. Often the granulomatous mass within the nares is sufficiently large and swollen to make it almost impossible to locate its attachment. However, it is well worth spending some time using vasoconstrictors and a probe to ascertain the point of attachment. In a case of any of these conditions, besides a careful physical examination in addition to the local examination, a microscopic examination should be carried out. This is especially imperative in order to determine the type of treatment to be used. A hemangioma of the nasal septum arises from the cartilaginous vomer junction or from the cartilage itself and more probably from embryonic rests rather than from stimulation of normal septal tissues.

Hemangiomas of the nasal septum fall into two classes, angioma cavernosum and angioma simplex. In addition, textbooks speak of "bleeding septal polyp." The latter probably would fall into one or the other of the two preceding histopathologic classes if carefully examined.

Rosenthal [1] cited an instance in which a bleeding tumor was removed by a cold snare from the right anterior inferior corner of the septum, leaving a smooth cartilage behind. It was a question whether it was a hemangioma or granulomatous tissue. However, the consensus favored the former. Dabney [2] reported a case in which a large cauliflower growth was attached just posterior to the nasolabial margin. The growth had perforated the nasal septum, filling the right naris and fitting into

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From the Department of Surgery of the University Hospitals and the Western Reserve University School of Medicine.

1. Rosenthal, Morris: Hemangioma of the Nose, Laryngoscope **35**:54, 1925.
2. Dabney, Virginius: Angioma of the Nasal Septum: Case Report, Ann. Otol., Rhin. & Laryng. **40**:611, 1931.

the perforation so snugly that inspection on the left revealed no septal defect. After removal of the growth with a Luc forceps and cauterization of the denuded surface of the pedicle three times with tri-chloracetic acid, only a small pealike body of fibrous tissue recurred. This was snared off later, with no return of any growth for four years. Callison [3] reported a case of bilateral polypoid hemangioma of the nasal septum in which smooth, pedunculated dark red excrescences, ranging in size from that of a pea down, occurred at the junction of the crest of the vomer and the triangular cartilage bilaterally. These bled on the slightest touch. Callison suggested that it would have been necessary to obliterate the tissue at the junction of the crest of the vomer and the triangular cartilage in order to effect a cure. Tilley [4] mentioned a "large angeioma" producing a swelling of the right side of the nose for eight months. Seven months after its removal, it rapidly recurred, so as to project from the nostril and bleed freely. Then it was radically removed, along with its pedicle attachment to the septal cartilage. The opinions of pathologists differed as to whether the growth was an angioma or a melanotic sarcoma but favored the first.

Treatment varies with individual surgeons, but on the whole it consists of four forms: cauterization, use of radium, electrodesiccation and excision. Since the most common sources of such growths are the anterior vascular portion of the septum, Kiesselbach's plexus and the region along the vomer cartilaginous junction, the base of the tumor is limited by the cartilage. Excision seems to be the most logical treatment, because then all factors are definitely under control and there is less likelihood of perforation. This is not the case with electrical, thermal or chemical agents. A simple resection, including the cartilage, is readily done without any perforation of the septum resulting. Since cartilage is relatively avascular, it affords a good substance for slow spread of growth and invasion. Therefore, most of the noninfectious granulomas arising over cartilage, if observed early, can be completely removed by including the base and underlying cartilage along with the tumor itself. This leaves the submucous tissues on the opposite side of the tumor viable and thus prevents a perforation of the septum. Even if the cartilage is not involved (this is likely to be the case), it is best to remove it at the same time as the granuloma. If this is not done, the cartilage usually dies and sloughs later if any large surface has been denuded on one side during such an operation.

In none of the three cases reported here has the growth shown any tendency to recur since the removal by surgical excision, as contrasted

3. Callison, James G.: Bilateral Polypoid Hemangioma of Nasal Septum, Laryngoscope **27**:33, 1917.

4. Tilley, Herbert: Large Angeioma of the Nasal Septum, Proc. Roy. Soc. Med. (Sect. Laryng.) **14**:34, 1920-1921.

with the regrowth in two of these cases previously when the growth had been simply removed with a cold snare.

REPORT OF CASES

CASE 1.—C. D., a white man of 26, was admitted to the outpatient department of the Lakeside Hospital on Dec. 27, 1929. He complained of having had frequent nosebleeds during the preceding six weeks.

The nasal septum was deflected to the left; the space was poor on the left. There was a tumor of the right side of the septum. The membranae tympani were normal. The tonsils were enlarged.

On December 28, with the area under local anesthesia induced by cocaine and epinephrine, the tumor was removed from the right side of the septum by means of a cold snare. On December 30 the operative site was touched with 10 per cent silver nitrate.

The results of histologic examination were reported as follows: "The tumor was represented by a small nodule covered by stratified epithelium, which was in places ulcerated and encrusted with fibrinopurulent exudate. The nodule was comprised of small blood vessels supported by a scanty fibrous stroma, sparsely infiltrated by exudative cells. The character and distribution of the vessels throughout the nodule would seem to indicate that the lesion is a vascular tumor rather than granulation tissue."

In February 1936 the patient reported that he had had no further trouble, and he refused to come in for an examination.

CASE 2.—B. L., a white man of 52, was admitted to the outpatient department of the Lakeside Hospital on Feb. 11, 1930, complaining of nasal obstruction of two months' duration and of occasional nosebleeds. Two days before examination, he had had a nosebleed which lasted one-half hour.

There was a soft pendulous mass in the right naris, completely blocking the passage. It bled profusely when it was touched with a probe. The left naris had fair space. The tonsils were hypertrophied. It was impossible to get a view of the nasopharynx.

Results of roentgen examination on February 13 by Dr. David Steel were reported as follows: "The right antrum is normal. The left antrum is larger than the right. The lateral wall is poorly defined."

On February 20 the tumor was removed from the right septum with a cold snare, with the area under cocaine and epinephrine anesthesia.

On May 10, 1930, the patient again complained of nasal obstruction. The growth had recurred on the right side, completely filling the naris. It bled freely when it was touched with a probe.

On May 17, 1930, a submucous resection of the base of the pedicle and about 1 cc. of the surrounding mucoperichondrium in mass was performed. This left a denuded cartilaginous area about 2.5 cm. in diameter covered with normal mucoperichondrium on the opposite side.

The results of histologic examination were reported as follows: "Most of the surface of this polypoid tumor was covered by an intact layer of stratified epithelium. The tumor had an abundant stroma, which was myomatous in places and supported many blood vessels, which were capillary in some places and cavernous in others. The impression was gained that there was active formation of new blood vessels and that this was a neoplastic process rather than the formation of new blood vessels in granulation tissue."

On May 20, 1930, there was much crusting at the operative site. On May 24 a piece of cartilage sloughed away in the area from which the tumor had been

removed. On June 3 the wound was granulating well, and there was considerable crusting. On June 21 and again on June 28 the granulations were cauterized with silver nitrate. On July 5 the granulations were smaller and were touched with a chromium tri-oxide bead. On July 12, August 2, August 16 and September 6 the wound was healing well, with a small number of granulations, which were cauterized each time with silver nitrate. On Sept. 20, 1930, the patient was discharged with the operative site completely healed and smooth.

Examination on Feb. 27, 1936, showed that there was no recurrence of the growth.

CASE 3.—B. L., a white married woman of 30, was examined on July 19, 1930. Since 1918 she had had recurrent growths from the right side of the nasal septum. Histologically the condition had been proved to be hemangioma. It had finally been treated with radium, and after that treatment the tumor was latent until her first pregnancy, which apparently stimulated its growth.. During this first pregnancy the growth was removed three times at Lakeside Hospital, with cautery, chromium tri-oxide bead and cold snare. The removal with the cold snare was carried out with the area under cocaine and epinephrine anesthesia during the last part of the pregnancy and about four months before the admission in July 1930. In less than two months after being snared off, the growth developed from a flat surface to a tumor so large as to protrude from the anterior naris.

The tonsils were small; there was no inflammation. The ears were normal. There was a growth extending from the middle of the nasal septum about opposite the middle turbinate bone and largely filling the right naris. Its pedicle was attached low down on the septal cartilage on the right side.

Under local anesthesia the tumor was snared off, so as to visualize its attachment. A circular area of perichondrium and the soft tissues beneath the attachment were elevated from the cartilage. Then through an incision on the opposite side of the septum, more anterior than the first, a similar circular area was elevated, leaving a disk of cartilage about 1 cm. in diameter surrounding the attachment of the pedicle, completely denuded of soft tissues. The mucous membrane for an area 1 cm. in diameter surrounding the pedicle, with the perichondrium and underlying cartilage, was removed. This left only the soft tissues of the opposite side of the septum from the tumor closing the defect.

The results of histologic examination were reported as follows: "Four surgical specimens were received for pathologic examination. The first was received on Dec. 17, 1929, the second on Feb. 8, 1930, the third on March 12, 1930, and the fourth on July 19, 1930. The first two specimens were similar, and each consisted of a polypoid tumor comprised of large and small, thin-walled and thick-walled blood vessels which approximated one another closely and which were supported by a scanty fibrous stroma. In a few areas undifferentiated masses of epithelium were observed. The superficial portions of the tumors were extensively ulcerated and encrusted with fibrinopurulent exudate. In the last two specimens the entire tissue was the seat of such extensive and severe inflammation that the identity of the structure was obscured and tumor could not be definitely recognized."

The convalescence was uneventful. The remaining perichondrium readily granulated over and became covered with epithelium. Except some crusting over the scarred area, there were no remaining symptoms. Since the operation there has not been any recurrence of the tumor or any bleeding. The patient has gone through two pregnancies without any recurrence of the tumor.

The histologic examinations were carried out by the department of pathology of the Lakeside Hospital.

Clinical Notes

ACUTE FULMINATING LARYNGOTRACHEOBRONCHITIS IN AN ADULT

DAVID DAVIS, M.D., Washington, D. C.

Acute fulminating laryngotracheobronchitis in children has been described by various writers. There is, however, a great paucity of literature concerning this condition in adults. The disease is usually seen in children from several months to about 9 years of age, the most frequent incidence being between 2 and 4 years. It is an extremely serious condition, owing to the obstruction of the respiratory tract by the swelling of the mucous membrane lining the trachea and bronchi and, lying on this swollen mucous membrane, a sticky, gummy exudate or secretion, which in turn causes more obstruction.

The typical case history is as follows: The child awakens at night with symptoms resembling those of spasmodic croup, with inspiratory dyspnea and hoarseness. Fever is usually present. Remedies such as those administered in cases of croup have practically no effect on the respiratory difficulty, and one thinks of diphtheria, or possibly a foreign body in the larynx, as a cause of the dyspnea. Antitoxin has been given by many physicians in cases such as this, without any beneficial effect. In order to rule out a diagnosis of diphtheria or foreign body, the larynx of the child is examined by direct laryngoscopy. Then the true nature of the disease presents itself. The larynx has a mottled reddish-gray appearance throughout; the vocal cords are injected and slightly thickened but move normally. The entire mucous membrane is velvety, but usually no exudate is found in the larynx itself. However, when the trachea is examined the cause of the dyspnea is apparent; the mucous membrane is thickened and velvety and is a dirty reddish gray, and lying on every part of it is a thick, tenacious secretion. The effect of the swollen mucous membrane and the secretion is to narrow the caliber of the lumen so that at times it is almost closed. The secretion is not always fluid or semifluid; often it is membranous and can be aspirated or even extracted with forceps as a cast of a part of the tracheobronchial tree. The removal of the secretion or membrane may give temporary relief, and the child may seem to be out of danger. However, in a short time the dyspnea reappears, with restlessness and cyanosis, and it becomes a question whether to perform tracheotomy or to insert an intubation tube in order to allow sufficient air to enter the lungs. Baum,[1] of Denver, advocates intubation first, but if dyspnea continues he advises tracheotomy. He stated that there is less tendency for crust to form in the tracheobronchial tree after intubation but that it is necessary for the child to be hospitalized where some one trained in inserting intubation tubes is always present. Richards [2] expressed the belief that tracheotomy is of more value as it is possible to aspirate the secretion more easily through a tracheotomy tube,

From the Department of Otolaryngology, George Washington University School of Medicine.

1. Baum, Harry L.: Acute Laryngotracheobronchitis, J. A. M. A. **91**:1097 (Oct. 13) 1928.

2. Richards, Lyman: Ann. Otol., Rhin. & Laryng. **42**:1014 (Dec.) 1933.

and, if necessary, any number of bronchoscopies can be done through the tracheotomy opening for purposes of aspiration without injury to the larynx. However, I believe that it is advisable to insert only the aspiration tube through the tracheotomy opening, as the passage of the bronchoscope often causes more edema and the production of more secretion in the trachea and bronchi, especially in young children and in infants. If the secretion is in the form of a membrane, it may be necessary to remove it with a forceps inserted through the bronchoscope; however, removal of the membranes through the aspiration tube is often satisfactory and should be tried first. Even with an intubation tube in the larynx, or with a tracheotomy opening, the patient often fails to obtain the necessary amount of air to maintain life and succumbs. There apparently is a marked toxic factor involved, for even when an open airway is maintained many of these children continue to become progressively worse until the fatal outcome. It would seem, with the dyspnea, restlessness and cyanosis, that the obstruction is at the laryngeal end of the tracheobronchial tree, but on inspection with the bronchoscope it is found that often the trachea is open but the bronchi are occluded by secretion and swollen mucous membranes. Aspiration of the secretion frequently gives the child a breathing space for a few hours, and with the milder type of involvement may be sufficient to save the child's life. Nevertheless, it soon becomes a contest between the aspiration and the formation of secretion, and with the fulminating type it is impossible to maintain an open airway, and the patient dies. Usually this takes place within a few days after the onset of the symptoms and even after the child has apparently improved several times, so that one must be careful in making the prognosis. Various other measures for relief from the dyspnea have proved worthless in most cases. For instance, the oxygen tent has failed to supply the necessary aeration of the lungs, so that the patient does not improve when this method of therapy is used. This may be due not only to obstruction to the entrance of oxygen to the lungs but to the fact that pneumonia so often is present. However, it is necessary to try all methods for getting oxygen into the lungs and for maintaining an open airway, so that repeated aspiration through the bronchoscope of the gluelike secretion from the trachea and bronchi, the oxygen tent, along with as much alkaline fluids as can be administered either by mouth or rectum or by hypodermoclysis, and the steam kettle must all be used if one hopes to keep the patient alive.

In almost all of the cases reported the offending organism was Streptococcus haemolyticus, Bacillus influenzae or Staphylococcus. In the case which I wish to report the culture and smears showed only staphylococci. The patient was an adult, but the character of the secretion found in the tracheobronchial tree was similar to that found in the trachea and bronchi of children, and the course of the disease was likewise one of rapid fatal termination. Gardner,[3] of Boston, in 1918 reported three cases of acute laryngotracheobronchitis in children in which pure cultures of Staphylococcus pyogenes-aureus was found, not only in the superficial exudate but in the deeper layers of the trachea and bronchi. He expressed the belief that the organism remains in the local lesions of the trachea and bronchi but that the severity of the disease is due to the absorption and distribution of toxins through the blood and lymph streams. All of his patients died. Lynch,[4] of New Orleans, in 1924 reported five cases of mild laryngotracheobronchitis in children in which there was a membranous formation in the trachea and bronchi. The offending organism was Staphylococcus albus. Tracheotomy was performed

3. Gardner, L. U.: Am. J. M. Sc. **155**:380 (March) 1918.
4. Lynch, R. C.: Laryngoscope **34**:753 (Oct.) 1924.

in four cases, and aspiration of the tracheobronchial tree was performed in all the cases. He advocated warm, moist air of even temperature, free purgation of the tracheobronchial tree with such drugs as ipecac and small doses of iodides not over 3 grains (0.18 Gm.); small doses of mild mercuric chloride and antimony and potassium tartrate or squill are to be relied on, coupled with mild nonirritating alkaline diuretics and intestinal flushing. He stated that intubation is unjustifiable and advocated tracheotomy. All of his patients recovered, but decannulation could not be done for from four to nine weeks. The course in his cases was nearly afebrile and apparently nontoxic, except locally, and no sequela resulted. He stated that antitoxin is worthless in these cases. Apparently the involvement in his series was of the milder type and the cases are not to be compared with the others reported in the literature.

REPORT OF A CASE

Mrs. E. S., aged 29, was admitted to the George Washington University Hospital on Dec. 28, 1934, with the following history: A sore throat developed three days before admission. The patient continued to get worse, and the day before admission she began to lose her voice. The night before admission she had difficulty in breathing owing to soreness and swelling in the throat. She was unable to give any further history owing to the loss of voice and soreness of the throat. She was dyspneic and cyanotic. The pulse was rapid, of good quality and regular; the blood pressure was 100 systolic and 70 diastolic. There was retraction of the sternum on inspiration, with the sides of the chest flaring widely. The temperature was 101 F. Examination of the sputum for Pneumococcus types I and II gave negative results. The examination of the blood was interesting. The white cells numbered 1,650, wih 26 per cent segmented forms, 13 per cent band forms, 15 per cent juvenile forms, 3 per cent myelocytes, 3 per cent monocytes and 40 per cent lymphocytes. The blood sugar content was 175 mg. per hundred cubic centimeters. The patient was placed in an oxygen tent but apparently was not improved by this therapy. Previous to this steam inhalations were given, with equally poor results.

Examination of the larynx with the mirror showed diffusely reddened vocal cords, which moved well on phonation and respiration. The posterior wall of the subglottic region protruded into the lumen of the trachea, so that the lumen could not be seen. Since it was not possible to determine the extent of the tracheal involvement, direct laryngoscopy and bronchoscopy were advised. Physical examination of the chest showed bronchopneumonia of the right lung. A roentgenogram showed some mottling throughout the entire right lung and in the middle portion of the left lung. This was interpreted to be bronchopneumonia but could possibly have been primary tuberculosis. Direct laryngoscopic and bronchoscopic examination showed the following picture: The larynx was diffusely injected and had a mottled reddish-gray appearance. The cords moved normally but were also red and slightly thickened. No membrane was seen. The trachea and bronchi, however, showed considerable pathologic change; the mucous membrane throughout was a mottled, reddish gray and very velvety. It looked like a cushion and lying on its entire surface was thick mucus, tinged with serosanguineous fluid. The lumen of the trachea and the bronchi was greatly diminished by the swollen mucous membrane and secretion. The smaller bronchi appeared almost entirely closed, and some thin serosanguineous secretion could be aspirated from them. The patient seemed to breathe better while the bronchoscope was in situ and following the aspiration. It was decided to perform a tracheotomy at once in order to aspirate the secretion more frequently; therefore, with the bronchoscope still in the trachea, the operation was carried out. The vessels of the neck were engorged to

several times their normal size but fortunately were not injured during the tracheotomy. The patient was returned to the ward and placed in the oxygen tent, but even after aspiration and tracheotomy she did not breathe much better, and her condition did not appear to be much changed. Suction through the tracheotomy tube with a rubber catheter and a curved metal catheter removed small amounts of serosanguineous secretion from the trachea and the bronchi, but with no relief from the dyspnea. Forced feeding of water, orange juice and milk was done, and epinephrine, caffeine sodiobenzoate, digifolin, insulin, dextrose and atropine were given by hypodermic injection. The temperature was 101 F. at the end of the bronchoscopy, but it gradually mounted to 104 F. In spite of aspiration and the administration of oxygen and medication, the patient died fourteen hours after tracheotomy.

Examination of the aspirated bronchial secretion showed almost a pure culture of Staph. aureus and an occasional streptococcus. No Klebs-Löffler bacilli were found. Thinking that the culture may have become contaminated, at autopsy a culture of the mucous membrane of the trachea and bronchi was made, and a pure culture of Staphylococcus aureus was obtained. Autopsy revealed considerable congestion of the larynx, trachea, bronchi and bronchioles, with acute inflammation and a deep red color. In the larynx were many small grayish patches, which were of diphtheritic nature. Both lungs showed many reddish areas of consolidation of varying sizes, in definite relationship to the small bronchi and bronchioles; that is, they surounded their terminal branches. This was a type of bronchopneumonia.

The anatomic diagnoses were (1) acute laryngotracheobronchitis, with secondary bilateral bronchopneumonia; (2) toxic myocarditis, and (3) toxic splenitis, hepatitis and nephrosis.

COMMENT

The fulminating character of this disease almost precludes any successful therapy. The course is so rapid, the toxicity so great and the respiratory obstruction so marked that the patient has little chance for a successful outcome. In the majority of the many cases in children reported in the literature there was a fatal outcome within a few days. Most of those children who lived had a milder type of infection, and probably their condition resembled that in cases which one observes but in which one seldom does more than to prescribe use of the steam kettle and a little ipecac. Obstruction of respiration apparently is not the sole cause of death, for in several cases a free airway was maintained, and the patients suddenly died while apparently improving. There is unquestionably a large toxic factor which overwhelms the whole body, as is shown in my case in which the white blood count was but 1,650, or it may be that the obstruction in the bronchi is so near the lungs and so widely disseminated that it cannot be relieved by such procedures as intubation, tracheotomy, bronchoscopy or suction or the use of an oxygen tent.

Although all cases of acute fulminating laryngotracheobronchitis which I have been able to find reported in the literature occurred in children under 9 years of age, the onset, symptoms, course, outcome, bacteriology and autopsy observations in my case show the condition to be the same, and therefore laryngotracheobronchitis should not be classed only as a disease of childhood.

CONCLUSIONS

Acute fulminating laryngotracheobronchitis is usually a rapidly fatal disease.

It is usually a disease of childhood but does occur in adults, as shown by the case reported here.

The offending organisms are Str. haemolyticus, Staph. aureus and the influenza bacillus.

The patient dies from suffocation and toxicity.

There is no specific remedy for this condition. A free airway and elimination of the toxins are necessary to maintain life.

The pathologic process is most marked in the larynx, trachea and bronchi and in the lungs surrounding the smaller bronchi and bronchioles.

The disease resembles spasmodic croup at the onset; a diagnosis of diphtheria and foreign body in the respiratory tract must be eliminated.

Bronchoscopic aspiration has proved helpful in maintaining an open airway in many cases.

Tracheotomy is more to be relied on than intubation for maintenance of an open airway.

1835 Eye Street, N. W.

A SIMPLE MANIKIN WITH AN ELECTRICALLY ACTUATED BRONCHUS FOR BRONCHOSCOPIC DEMONSTRATIONS

R. S. Rosedale, M.D., M.Sc. (Med.); D. R. McKay, M.D.; B. R. Stephenson, Ph.D., and H. A. Stevens, Buffalo

Recently we wished to construct a replica of a bronchus of the first order compromised by carcinoma. This was to form part of an educational exhibit of bronchogenic carcinomas at the annual session of the American Medical Association. As we did not wish to resort to the frequently used moulage process, we fashioned a bronchus in rubber, molded into it an irregular mass resembling carcinoma and painted the whole model with appropriate colors. A machine was then constructed which would make the bronchus expand and contract as it does in life, so that a view through a bronchoscope inserted into the bronchus through a dummy head would give a realistic illusion of seeing a bronchogenic carcinoma in a living patient. This was enhanced perhaps by an inspiratory and expiratory surge of 500 cc. of air, representing tidal air, eighteen times a minute.

A number of bronchoscopists who viewed this apparatus suggested that it might be used as a teaching adjunct for demonstrating to practitioners and medical students the value of direct examination of the tracheobronchial tree and to impress them with the importance of this procedure in the diagnosis of certain obscure conditions. It could perhaps be utilized also in demonstrating to the same groups the removal of foreign bodies. We therefore wish to present it as a teaching aid in this field.

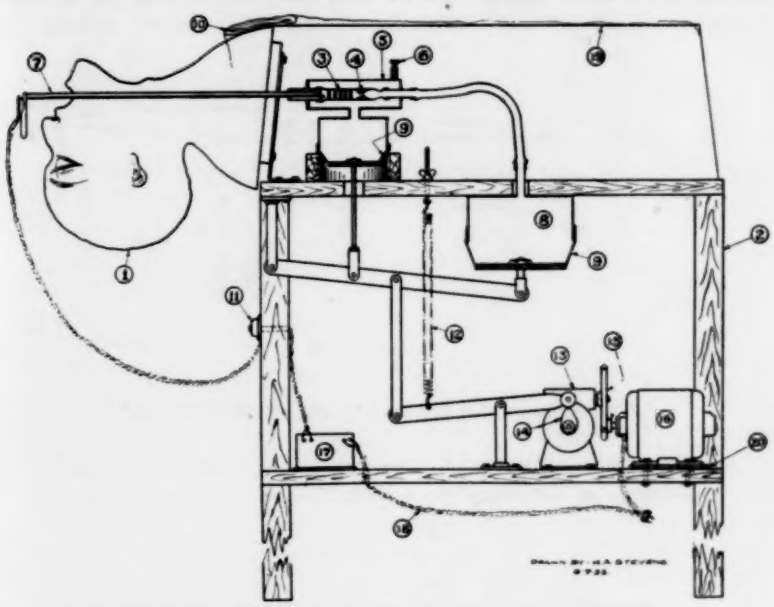
DESCRIPTION OF APPARATUS

The purpose of the mechanical part of the breathing model was twofold: first, to make the artificial bronchus expand and contract and, second, to make a gush of air pass in and out of the mouth of the model in a manner simulating breathing.

From the Departments of Pathology, Medicine and Physics, the Buffalo City Hospital and the University of Buffalo School of Medicine.

This was accomplished by building two expansion chambers operated by means of motor-driven diaphragms, acting as a bellows.

In the figure is shown a one-fourth horse power washing machine motor (*16*) which is connected by a belt (*15*) to a reducing gear (*13*) which gives eighteen revolutions per minute to the slow speed shaft. On the shaft of the reducing gear is attached a cam (*14*) so constructed that the levers attached to the expansion chambers (*8*) are forced down slowly and then return to the highest position rapidly. The effect of this cam is to produce a slow intake of air, simulating inspiration, and a rapid expulsion of air, simulating expiration. The diaphragms (*9*) in this model were made of sheet rubber, held on with heavy rubber bands. These diaphragms would be more durable if they were of leather and held on with a metal band. A heavy coil-spring (*12*) holds the wheel at the end of the



A simple manikin with a mechanically actuated bronchus.

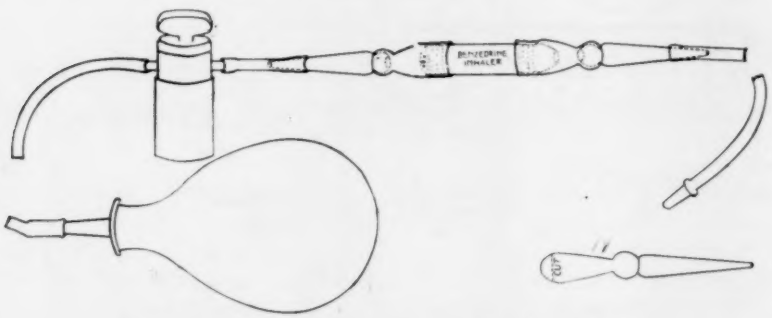
lever firmly against the cam (*14*), in order to prevent undue noise and backlash. Within the metal tube (*5*) the rubber bronchus (*3*) is sealed. The molded "cartilaginous" rings (*3*) and tumor (*4*) of the rubber bronchus are rubber-cemented to the inside of the bronchus in order to simulate a tumor. A vent (*6*) permits the entrance of the correct amount of air into the chamber (*5*) so as to obtain the desired amount of expansion and contraction when the diaphragm moves in and out.

The cardboard cover (*19*) for the model is covered with a sheet or some other drape. A toy transformer (*17*) illuminates the bronchoscopic lamp through the push button (*11*). The plaster cast of the bust (*1*) is made from a body in the autopsy room. The bust can be coated with ordinary house paint of appropriate color. The whole unit is mounted on a stand (*2*), measuring 24 by 26 inches (61 by 66 cm.) on top and 41 inches (104 cm.) in height.

A SIMPLE METHOD OF INTRODUCING BENZEDRINE VAPORS INTO THE EUSTACHIAN TUBE

JAROSLAV H. HULKA, M.D., LONG ISLAND CITY, N. Y.

Benzedrine (racemic benzylmethyl carbinamine) is a volatile substance which has a vasoconstrictory effect, similar to that of ephedrine. The "benzedrine inhaler" (accepted by the Council on Pharmacy and Chemistry of the American Medical Association) can be easily attached to the Dench vaporizer by means of two medicine droppers, as illustrated in the accompanying diagram. Insufflation of benzedrine vapor through a eustachian catheter is especially useful previous to the passage of eustachian bougies. I have also observed that the insufflation of benzedrine vapor relieved tinnitus in several cases of acute nonsuppurative



Benzedrine inhaler attached to the Dench vaporizer.

inflammation of the middle ear. So far, I have not observed any symptoms of labyrinthine irritation following the introduction of this potent drug into the middle ear.

A HISTORICAL NOTE REGARDING PETROSITIS

R. WALLACE TEED, M.D., ANN ARBOR, MICH.

The great interest shown in infections of the petrous bone during the last six years has been construed by some to indicate that knowledge of these conditions is essentially new.

A review of the literature reveals that the signs and symptoms were well recognized and the methods of treatment were devised by 1904. Furthermore, some of the old masters, like Tröltsch, were acquainted with the syndrome thirty-five years before that.

What was probably the earliest study of petrositis or at least one of the earliest, was made by Edward Cock in 1838. In his article entitled "Contribution to the Pathology of Congenital Deafness" [1] he reported pathologic studies of four

1. Cock, E., Guy's Hosp. Rep. **3**:289, 1838.

patients with congenital deafness showing anomalous labyrinths. No histories were given, and the protocols were limited largely to the condition in the ears. In case 4 the right tympanum was filled with mucopurulent fluid. "The petrous bone, more especially in the neighborhood of the tympanum, was unusually loose and cellular, presenting numerous irregular cavities, which apparently communicated with the tympanum and were filled with the same mucopurulent fluid; they seemed a sort of extension of the mastoid cells."

Cock apparently did not appreciate the significance of this finding, but he must be credited at least with acute powers of observation.

410 Highland Road.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

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OTTAWA, CANADA

Fewer articles dealing with otosclerosis and pathologic changes in the labyrinth appeared in the literature in 1935 than in most other years. The most important paper on the pathology of otosclerosis is that of Greifenstein.[1] In this study he describes two otosclerotic bones to which there had been operative trauma with accompanying regenerative processes.

In the study of the pathologic changes in the bones he emphasizes the importance of polarized light in showing up the fibrils. He employs a special filter (*Gipsblättchen rot 10*), the interposition of which shows fibrils in the appropriate axis as blue or yellow while the ground is red.

The otosclerotic bones studied were from a man who had been operated on by the translabyrinthine route for supposed tumor of the acoustic nerve and who died twenty-seven days later. There were otosclerotic changes on both sides, with varied histologic pictures. In the region of the semicircular canals there were many so-called mantle formations which were sharply defined from the rest of the bone. These mantles spread as finger-like processes into the surrounding bone. Osteoclasts were seen at the advancing tips. Deposition of new blue bone appeared to occur just as fast as the absorption of the normal bone progressed. This blue bone was poor in fibrils but rich in cement substance. The fibrils were not arranged in parallel bundles but formed a weblike structure. In the region of the canals the mantles were so numerous as to coalesce. In places sequestrums of old bone were enclosed by new blue bone. These blue mantles are later absorbed by osteoclasts and replaced by red bone with numerous fibrils, such as is seen in older parts of the focus. This, in turn, is sometimes replaced, and there is a breccia of mixed elements.

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1. Greifenstein, A.: Vergleichende Untersuchungen zur Histologie der Otosklerose, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:14, 1935.

Greifenstein concludes from his own observations that the otosclerotic bone is a new formation following previous absorption.

The second section of Greifenstein's paper is devoted to osseous regeneration and otosclerosis. It has been stated that otosclerotic bone resembles callus. Callus is formed from specific germ tissue with weblike bony tissue which is much richer in fibrils than otosclerotic bone, and the typical blue staining is absent. If the blood supply is good and the germ tissue is active, callus formation is quickly completed, and the bone is then replaced by vascular marrow spaces, leading in turn to the formation of lamellar bone. Weblike bone is seen only in localized areas and for a short time. In the formation of the weblike bony trabeculae in callus there is a distinct tendency for the fibrils to be arranged in definite directions, as contrasted with the irregularity in young otosclerotic bone.

Greifenstein studied the edges of trephine openings in the skull. The most satisfactory pictures were obtained in a case in which the skull was opened twenty-seven days before. There was no evidence of regeneration of bone opposite the outer and inner tables, but where the diploe had been opened there was active growth of callus. Spongy blue bone was seen with osteoblasts at the edges. It was separated from the old bone by a cement line. The old bone showed changes in the form of blue mantles, which appeared in vascular channels for some distance from the regenerating edge. This callus was extraordinarily poor in fibrils.

In the otosclerotic specimens already mentioned the bone in the region of the middle coil of the cochlea had been replaced by otosclerotic bone. This part of the bone had been opened with a chisel during the operation twenty-seven days previously. The defect had been completely closed by a new network of spongy bone, which joined with the otosclerotic bone and spread into the cavity of the labyrinth. The new bone was stained red-violet in contrast to the dark blue of the otosclerotic bone. In many places there was no sharp line of demarcation, the two types of bone merging into each other. The fibrils became more numerous, and they were arranged in bundles in passing from otosclerotic bone to callus. In other areas where there were necrotic fragments of the blue bone these were sharply separated from the callus. In one place, where an otosclerotic focus had been injured, there was formation of ordinary callus in the middle of the focus. Where the chisel had cut into the older parts of the focus the callus was separated from the old bone by a distinct cement line. The callus here was poor in fibrils, and it stained blue. In all the areas the blood vessels were numerous and distended. In one spot, where a crack extended into blue young otosclerotic bone, there was an area of absorption with new pink callus in it.

In summing up this phase of his study Greifenstein points out that primitive bone with scanty irregularly arranged fibrils, rich in cement substance and staining blue as in otosclerosis, can occur under normal circumstances as callus. The necessary conditions for its occurrence are unknown, but it develops in places where regeneration is slow. There is enough resemblance between callus and young otosclerotic bone to suggest that the focus is a regenerative new formation occurring after previous absorption. The power of regeneration of the three layers of the labyrinthine capsule is dependent on the presence of specific germ plasm in the marrow spaces. It is absent in the enchondral bone and endosteal bone, where there are no marrow spaces, but it is present in the periosteal bone, where there are marrow spaces. The callus is derived from the osteoblasts of the marrow.

The demonstration of active callus formation when an otosclerotic focus is injured shows definite proof of the osteogenic potency of the cells of the focus. Injury to the older parts of the focus causes a variable amount of callus to develop, depending on the quantity of osteoblastic marrow present.

Greifenstein devotes a section of his paper to the histologic picture of osteoma. Osteogenic neoplasms of an inflammatory nature in the sinuses are composed of spongy or compact lamellar bone. Exostoses in the ear show no web bone. Their bony changes are due to osteoclastic activity, followed by the deposition of new lamellar bone.

On the other hand, an osteoma of the frontal sinuses shows a complicated structure. Here and there strand bone (*Strähnenknochen*) is observed around the vessels. The bone is closely similar to the periosteal layer of the labyrinth. The unusual richness in fibers of the eburnated bone resembles the labyrinthine capsule. There are no cement lines between the strand bone and the lamellar bone, and the transition between them is gradual. Inside the osteoma florid changes, with the typical picture of osteodystrophia fibrosa, are noted. The compact layer changes from lamellar to web bone, or breccia, with characteristic blue mantles. The microscopic picture of the osseous changes in an osteoma is completely identical with that observed in otosclerosis.

In experimentally produced zones of activity (*Umbauzonen*) in the long bones of dogs the fibrils are fewer and stain less deeply than is the case in otosclerosis. In certain areas pictures somewhat resembling those typical of otosclerosis are seen with osteodystrophia fibrosa, web bone, breccia, blue mantles, etc. Intravital cracks also are seen.

Greifenstein goes into the question of so-called spontaneous fractures. He examined cracks produced by operative trauma and observed that in some parts there was definite callus, while in other parts, where the bone was dense, the fissure was empty. The changes depended on the type of bone traversed. Some of the operative cracks passed

through an otosclerotic focus. Here they were filled with cells and some pink-staining callus, which tends to prove the regenerative power of otosclerotic bone.

Fractures were seen also on the side not operated on in the same patient. They contained cells which stained well and were evidently derived from the otosclerotic focus. Greifenstein believes that these occurred intra vitam. Spontaneous fractures were seen in experimentally produced zones of activity in the ulna of the dog. They were never seen in web bone, only in compact bone. The fissures sometimes contained blue-staining material. There was no blood in them.

Otosclerosis is no unique condition, as essentially similar pictures are seen in cases in which there is regeneration of bone and in the conditions included under the name osteodystrophia fibrosa. The young otosclerotic focus is a regenerative new formation after previous absorption. With regard to the cause of otosclerosis, Greifenstein points out that the widely filled blood vessels, which Wittmaack made the focal point of his theory, are seen in the most varied bone activities, especially in osteomas and in experimental zones of activity (*Umbauzonen*). There are close resemblances between otosclerosis and functional bone activities. Not all labyrinthine fissures are artefacts. Known experimental fractures in animals agree in their characteristics with spontaneous labyrinthine fractures. Mayer's theory of mechanical stresses as a cause of otosclerosis receives some support from this piece of work.

Helmuth Richter [2] gives the details of a case of otosclerosis associated with chronic suppuration of the middle ear. This was a case of otitis media with a granuloma in which a radical mastoid operation had been performed. Death occurred three days later. The side not operated on showed otosclerotic foci on the roof of the carotid canal, below the cochlea, on the promontory and near the bulb. On the side operated on there was a focus below the cochlea. The stapes, which was otosclerotic, was displaced into the vestibule. The middle ear was full of angiomatous granulation tissue, which extended into the attic and down to the jugular bulb under the labyrinth. There was pus in the inner ear and in the internal meatus. The danger of curetting the inner ear was pointed out. The stapes had probably been more easily displaced because it was brittle and the annular ligament was destroyed.

Oppikofer [3] describes the changes in the ear in a case of Niemann-Pick's disease. This disease is one of the lipoidoses in which there is a disturbance of lipoid metabolism, with the deposit of fatty material

2. Richter, Helmuth: Beiträge zur pathologischen Anatomie des Ohres, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:181, 1935.

3. Oppikofer, E.: Histologische Ohrveränderungen bei Niemann-Pickscher Krankheit, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:77, 1935.

in the tissues of various organs, such as the spleen, liver, bone marrow and lymph glands. There are three varieties of lipoidosis:

1. Gaucher's disease (cerebroside lipoidosis).
2. Niemann-Pick's disease (phosphatide lipoidosis).
3. Christian-Schüller's disease (cholesterol lipoidosis).

The clinical course of the disease is characterized by enlargement of the spleen and liver. The characteristic foam cells of Gaucher's disease arise from reticulum cells; those of Niemann-Pick's disease, from epithelial cells.

One case of Gaucher's disease has been described by Brunner and one of Niemann-Pick's disease by Druss. The Niemann-Pick cells are present in the epithelium of the tube, tympanum, antrum and cells and in the marrow and between the bundles of the acoustic nerve.

Oppikofer's case is that of a boy of 27 months who had developed normally until the age of 10 months, when the spleen and liver began to enlarge. The sight gradually diminished until there was total blindness, with a red macula. The boy became deaf or at least very hard of hearing and died of pneumonia. The temporal bones were studied and found to be symmetrically affected. Foam cells were observed in many places. They were large cells, 22 microns in diameter, with vacuoles and eccentric nuclei. Niemann-Pick cells were present under the skin of the external canal and drum. The middle ear showed inflammatory changes, with infiltration with round cells and some Niemann-Pick cells. Some of the foam cells were seen in the exudate also as well as in the marrow spaces of the ossicles and around the labyrinthine capsule. There were none in the enchondral bone. A few were seen in the inner ear, in the spiral lamina and under the two maculae. Otherwise, the inner ear appeared normal. The deafness was probably due to central changes.

A good deal of attention has been devoted to the significance of the fissula ante fenestram of late years with reference to otosclerosis, and the paper by Anson and Martin [4] is timely. They have made a detailed study of the fissula ante fenestram from serial sections and reconstructions of forty specimens. From their work it is realized what an extraordinary degree of variation this structure shows with regard to size and shape. The fissula is well established in a 14 week embryo. It contains primitive areolar tissue and is surrounded by cartilage. This cartilage persists throughout life in many cases and is most commonly seen at the vestibular end. It is continuous with the cartilage lining of the vestibular window. There is usually cartilage somewhere in the cleft,

4. Anson, Barry J., and Martin, J., Jr.: The Fissula ante Fenestram: Its Form and Contents in Early Life, Arch. Otolaryng. **21**:303 (March) 1935.

generally lining it but sometimes as a distinct mass. Nodules of immature cartilage may be present in the fissula itself or as an appendage to it.

With regard to the form of the fissula, it is more or less right angled, with a short tympanic end running medially and a longer and flatter vestibular end running posteriorly from it. The tympanic end is small; it is usually situated above the level of the vestibular end and may open into the semicanal of the tensor tympani. The vestibular end is usually a deep slit.

Bast [5] describes the blood supply of the otic capsule of a 150 mm. (crown-rump-length) human fetus of 18½ weeks. The capsule at this stage is partly ossified and partly cartilaginous. In brief, the arteries to the capsule are (1) the inferior tympanic branch of the ascending pharyngeal artery, (2) the posterior meningeal branch of the occipital artery, (3) the recurrent mastoid branch of the occipital artery, (4) the stylomastoid banch of the posterior auricular artery, (5) the accessory stylomastoid branch of the posterior auricular artery and (6) the subarcuate artery from the internal auditory artery or from the basilar artery.

With the exception of the subarcuate artery, all are derived from the external carotid artery. The inferior tympanic artery seems to constitute the main blood supply to the inner tympanic wall, but it anastomoses with most of the other vessels in the neighborhood.

Hagens,[6] writing on the pathology of otosclerosis from a study of five bones from four patients, discusses Gray's report of degenerative changes in the cochlear nerve and considers that they are open to question. He states that the neurokeratin filament is not normally distinguishable in the cochlear nerve, but it is distinguishable in the vestibular nerve. He was unable to see in his specimens any of the changes described by Gray. In two of his cases the condition was associated with chronic suppuration of the middle ear. Some of the specimens showed osteoblasts and osteoclasts, while others did not. The fissula ante fenestram was obscured in some of the specimens by the otosclerotic focus. Hagens concludes that no one theory of etiology is yet adequate to explain the condition.

Torrini [7] contributes a paper on otosclerosis. He reviews the histologic changes and the various theories as to the origin of otosclerosis. He carried out a series of experiments on male and female guinea-pigs

5. Bast, T. H.: The Blood Supply of the Otic Capsule of a 150-Mm. (C. R.) Human Fetus, Anat. Rec. **48**:141, 1931.

6. Hagens, E. W.: Pathology of Otosclerosis, with Report of Cases, Arch. Otolaryng. **21**:297 (March) 1935.

7. Torrini, G., Jr.: Contributo alle conoscenze sulla etiologia dell'otosclerosi, Valsalva **10**:347, 1934.

of ages corresponding to the period of onset of otosclerosis in man. He kept two animals as controls. In some cases one ovary or one testicle was removed, and in others bilateral oophorectomy or orchidectomy was carried out. The temporal bones were later examined, and not the slightest change was observed. Torrini concludes from this that partial or total loss of function of the gonads is at least not one of the important causes of otosclerosis.

Some years ago Gray suggested the term otosclerosis paradoxica for the condition in those cases in which the patient hears better under circumstances in which one would expect the hearing to be poorer. Ashcroft [8] reports two such cases. The first patient, a woman of 30, was deaf in both ears, and the deafness had become worse in the left ear since pregnancy four years before. Paracusis was present. The hearing was improved when the patient was excited or had taken alcohol. Conversational voice was heard at 6 inches (15 cm.) with the right ear and at 18 inches (46 cm.) with the left ear. The patient was seen again when she had been pregnant for seven months and at this time the hearing was markedly improved. Conversational voice was heard at 3 or 4 feet (92 or 122 cm.). She was seen again about three weeks after confinement, and she stated that she had become stone deaf. The hearing had been improved in the last weeks of pregnancy. Tests showed that conversational voice was heard hardly at all.

The second case was that of a woman of 45 with extreme deafness, which had begun when she was 20. She had heard much better during the last month of each of two pregnancies, but the improvement disappeared after confinement.

TREATMENT OF OTOSCLEROSIS

Gray [9] has elaborated a system of treatment based on his theory that otosclerosis is due to the failure of the vasomotor mechanism of the ear. Years ago he noticed that thyroid substance or its extract when applied to the nasal mucous membrane produces hyperemia which lasts for a considerable time. He conceived the idea of using a similar substance locally in the middle ear in order to produce hyperemia there, especially as improvement in hearing results in otosclerosis paradoxica when the blood supply is increased. He explains that he does not use thyroid with the idea of remedying any supposed thyroid deficiency.

Thyroxin, the active principle of the thyroid secretion, is used. It can be obtained in tablets of from $\frac{1}{64}$ to $\frac{1}{640}$ grain (0.001 to 0.0001

8. Ashcroft, Dudley W.: Two Cases of Otosclerosis Paradoxica, J. Laryng. & Otol. **50**:268, 1935.

9. Gray, A. A.: The Treatment of Otosclerotic and Similar Types of Deafness by the Local Application of Thyroxine, J. Laryng. & Otol. **50**:729, 1935.

Gm.). Gray usually employs $\frac{1}{128}$ grain (0.0005 Gm.). The canal is sterilized with alcohol. The membrane of the drum is anesthetized with the author's solution of cocaine in aniline. Care must be taken to wipe the skin free from aniline, as aniline is absorbed through the skin and is very toxic. A solution of $\frac{1}{128}$ grain (0.0005 Gm.) is then made in 4 minims (0.25 cc.) of distilled water. It is injected through the membrane on a level with the tip of the malleus about half-way to the posterior edge. The needle must be long and slender, and the point should be beveled short. Immediately after the injection the patient's head is tilted back so that the fluid flows to the region of the round window and also so that it will not escape down the eustachian tube. One injection is given weekly into alternate ears. Not more than from 4 to 6 injections are given. Gray has treated fourteen patients, and in seven the condition was somewhat improved though not strikingly. The cases are reported in detail.

Regardless of the correctness of Gray's theory of the cause of otosclerosis, his treatment does not seem to me to be very logical. It is conceivable that it might have some effect on the bony otosclerotic focus but hardly on the auditory nerve. I am not one of those who believe that the presence of a bony change in the capsule is necessarily a cause of deafness, especially as many specimens examined histologically have been discovered accidentally to contain bony changes in the absence of previous deafness. If, as Gray contends, there are changes in the auditory nerve with degeneration of the nerve sheaths, it is hardly likely that any vasodilator applied to the middle ear would have any effect on the auditory artery which springs from a totally different part of the arterial system and does not communicate, as far as is known, with the blood vessels of the capsule or of the middle ear.

Fraser,[10] in a paper read at a meeting of the Scottish Otological and Laryngological Society in 1934, reports on the result of an operation undertaken some years previously on a patient with otosclerosis. The patient, a woman, was first seen on March 5, 1917, with deafness of four years' duration. She could hear a raised voice at 6 inches (15 cm.) with the left ear and conversational voice at 2 feet (60 cm.) with the right ear. C-32 and C-64 forks were not heard. A C-128 fork was heard with the right but not with the left ear. The Rinne test gave negative results for both ears. The upper tone limit was 12,000 vibrations. Radical mastoidectomy was performed on the left (worse) ear on June 28. The bony capsule of the lateral semicircular canal was uncapped, and a skin graft was applied. On November 8 the hearing was better. A C-64 fork and conversational voice at 3 feet (92 cm.)

10. Fraser, J. S.: Bilateral Otosclerosis: Radical Mastoid Operation on Worse Hearing Ear; Bony Cap of Lateral Canal Removed and Skin Graft Applied, J. Laryng. & Otol. **50**:700, 1935.

could be heard with the left ear. The operative cavity was moist, but no pus was present. The patient was seen again on Oct. 8, 1934, and conversational voice was heard at 6 or 7 feet (185 to 215 cm.). The fistula symptom was present.

Ballenger and Patterson [11] in an interesting paper refer to the earlier efforts of Urbantschitsch in auditory reeducation as well as the method of Zünd-Burguet, advocated by Cathcart, Yearsley and others. In order to ascertain the value, if any, of such methods, they employed an instrument constructed on the principle of the radio which could emit sounds of varying frequency and intensity. They gave 777 treatments in a period of ten months to both ears of twenty-five partially deaf adults. An average of 31 treatments were given to each patient. Fourteen of them suffered from nerve deafness, three from otosclerosis and the rest from various other forms of chronic deafness.

After the treatments had been given there was an average loss of 3¼ sensation units over the entire scale, as shown by the audiometer. Five patients showed a slight improvement, of from 2 to 12 units. The patients who showed the most improvement were those with chronic middle ear deafness. Ten patients reported for a check-up from four to ten weeks after completion of the treatment, and no change was observed.

PATHOLOGY OF THE INNER EAR

A. Brownlie Smith [12] reports on a case of influenzal labyrinthitis without otitis media. A child of 5 months had pneumonia followed by empyema. Puncture was carried out, and pus was withdrawn. The next day signs of meningitis developed, and death ensued in three days. At autopsy general meningitis was observed, and Bacillus influenzae was recovered from the pleural pus and the brain. The left temporal bone was removed and sectioned. The middle ear was normal. The inner ear contained pus, the leukocytes being mainly polymorphonuclears. They were for the most part seen in the perilymphatic spaces. The pus was traced along the acoustic nerve to the inner ear. The pneumonia evidently was the primary infection and was followed by blood-borne infection of the meninges and labyrinth.

Popow [13] describes a case of progressive nerve deafness with a strong hereditary influence. A man aged 30 heard well until the age of 10 years, when a gradual loss of hearing began. In 1926 he had malaria,

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11. Ballenger, H. C., and Patterson, B. A.: "Aural" or "Acoustic" Method of Treating Deafness, Arch. Otolaryng. **22**:410 (Oct.) 1935.

12. Smith, A. Brownlie: Influenzal Labyrinthitis Without Suppurative Otitis Media, J. Laryng. & Otol. **50**:263, 1935.

13. Popow, N. A.: Hereditäre Atrophie der Hörnerven, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:407, 1935.

for which quinine was given in large doses. In 1929 he contracted syphilis, and in 1931 he again had malaria, accompanied with total deafness. Later the hearing improved slightly. Examination showed that there was severe nerve deafness and that the vestibular apparatus reacted less than normally. The family history was bad as regards both deafness and alcoholism. The patient's father was deaf and was an alcoholic addict. The grandmother was totally deaf; eleven members of the patient's immediate family were deaf, one or two being deaf-mutes. In this case Popow considers that a developmental defect of the cochlea can be ruled out, as the patient heard well in early life. The condition must have been due to degeneration of the acoustic nerves. Familial neurodystrophies show up much in this way.

Bozzi [14] made postmortem studies of the internal auditory arteries of two hundred patients, mostly over 50. The vessels were studied in their extra-osseous course. Arteriosclerosis was observed in 29.7 per cent. This is practically a repetition of the work done by Wangemann in 1932 and reported in these ARCHIVES in June 1933.[15]

Some experimental work on the effect of quinine on the auditory nerve has been carried out by Seiferth.[16] Large toxic doses of quinine were given subcutaneously to a series of dogs. No effect was observed on the inner ear during either acute or chronic intoxication. In rabbits during acute poisoning nystagmus could be elicited by a change of position. The nystagmus was of a mixed type. Vertical nystagmus also was seen. Normal reactions to turning and the caloric tests were found, and from this it was inferred that the nystagmus must be of central origin. The picture of toxic neuritis due to quinine is dominated by inflammatory changes around the blood vessels. The pure degenerative toxic form of neuritis is observed only in the presence of a general debilitated state, such as diabetes, tuberculosis and chronic renal disease.

Bast [17] in 1928 described a hitherto unrecognized structure which he called the utriculo-endolymphatic valve. It consists of a valvelike opening between the Y-shaped end of the endolymphatic aqueduct and the utricle. There is a wide slitlike opening with a rounded end of cellular connective tissue covered by columnar epithelium. This valve probably allows slow diffusion to take place from the utricle to the

14. Bozzi, E.: L'arteria uditiva interna nei rapporti con l'aterosclerosi, Arch. ital. di otol. **45**:100, 1934.

15. Dickie, J. K. M.: Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Inner Ear, Arch. Otolaryng. **17**:806 (June) 1933.

16. Seiferth, L. B.: Ueber die Wirkung des Chinins auf das Gehörorgan und den Vestibularapparat, zugleich ein Beitrag zum experimentellen Lagenystagmus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **37**:367, 1935.

17. Bast, T. H.: The Utriculo-Endolymphatic Valve, Anat. Rec. **40**:1, 1928.

saccule, but it would close if there were a sudden loss of pressure in the saccule. Hoffman and Bast [18] followed up this observation by studying the labyrinths of a number of common mammals of different species. They observed that the utriculo-endolymphatic valve was constantly present though varying slightly in shape.

In 1934 Bast [19] reported finding two cases of rupture of the saccule in which the utriculo-endolymphatic valve was firmly closed. The ruptures had occurred intra vitam, as was shown by the growth of epithelium over the edges. Bast points out that clinical cases have been described in which there was a loss of cochlear function while vestibular function remained, and he suggests this as a possible explanation.

G. E. Shambaugh Jr.[20] reports on three patients with Ménière's syndrome who showed marked nerve deafness and diplacusis. Tones were heard differently in the two ears. The author explains the symptoms on the hypothesis of the presence of an exudate on the basilar membrane on one side which weights it down and alters its response to sound waves. Two of the patients had infected tonsils; after tonsillectomy the vertigo and diplacusis disappeared, and the hearing improved. The other patient had a prostatic infection; after suitable treatment the hearing of this patient also improved.

OBJECTIVE TINNITUS

Viole and Love [21] report a case of subjective and objective tinnitus probably due to cerebral aneurysm. Cerebral aneurysm is commonest in the middle cerebral artery and is more frequently present on the left side. Objective tinnitus aurium is of two kinds: (1) a clicking sound due to chronic spasm of the tensor palati muscle and (2) a hum or murmur synchronous with the pulse. The hum or murmur is usually due to an aneurysm but sometimes to an abnormal jugular bulb. Iglauer reported a case of objective tinnitus in which the patient was under observation for ten years. The sound disappeared on pressure over the carotid artery or on rotation of the head to the affected side.

Viole and Love's case is that of a woman of 53 who had had deafness and tinnitus in the right ear for over a year. She had also pain under the left eye and a discharge from the nose. Many intranasal

18. Hoffman, E. F., and Bast, T. H.: A Comparative Study of the "Utriculo-Endolymphatic Valve" in Some of the Common Mammals, Anat. Rec. **46**:333, 1930.

19. Bast, T. H.: Function of the Utriculo-Endolymphatic Valve: Two Cases of Ruptured Saccules in Children, Arch. Otolaryng. **19**:537 (May) 1934.

20. Shambaugh, G. E., Jr.: Syndrome of Diplacusis and Nerve Deafness for Low Tones, Arch. Otolaryng. **21**:694 (June) 1935.

21. Viole, Pierre, and Love, A. A.: Objective and Subjective Tinnitus Aurium of Vascular Origin, Ann. Otol., Rhin. & Laryng. **44**:418, 1935.

operations had been performed. Nine months previous to examination by Viole and Love she had suddenly noticed a pounding noise in the right ear, which was controlled by pressure on the right side of the neck. The noise had been constant ever since and sounded like swishing water. It was loud enough to interfere with the patient's sleep, and it could be heard by the examiner when a stethoscope was applied to the side of the head. There was concentric impairment of vision and a 20 per cent decrease in hearing on the right side, though the vestibular system was normal. Nothing abnormal was seen in roentgenograms. The authors concluded that there was probably an aneurysm of the posterior inferior cerebellar artery at its origin from the vertebral artery.

Two cases of objective noises in the ear are described by Gauger.[22] The first was a case of traumatic aneurysm of the vertebral artery. The internal jugular vein and all its branches were greatly enlarged. There was no communication with the carotid artery. The common facial vein was tied and divided, and the internal jugular vein was followed up to the jugular foramen, where it was adherent to the carotid artery. Severe bleeding was encountered; the carotid artery and the jugular vein were tied, and the bleeding point was encircled. The noise did not entirely disappear but was much weaker.

The second patient had a greatly enlarged vessel behind the mastoid which looked like a vein but pulsated strongly. A tortuous artery was located below it. The whole vascular mass was removed, and the emissary vein was plugged. The noise was then no longer objectively perceived. Following this the hearing was improved, but there was still some tinnitus.

A fairly comprehensive review of the literature on tinnitus is contributed by Gustl Weber.[23] The causes of objective tinnitus can be grouped into changes of the blood vessels and muscular contractions. In some of the cases of tinnitus due to muscle spasm the palate could be seen to jerk at the same time that the noise was heard.

BEETHOVEN'S DEAFNESS

Beethoven's deafness has been a subject of interest and speculation ever since the great master's death. A few years ago a paper on this subject was reviewed in these pages. Ira Frank[24] contributes another article. He points out that Beethoven had had very poor health all his life and had suffered from many severe attacks of intestinal disorders.

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22. Gauger, K.: Ueber objektive Ohrgeräusche, München. med. Wchnschr. **82**:567, 1935.

23. Weber, Gustl: Subjektive und objektive Ohrgeräusche, Internat. Zentralbl. f. Ohrenh. **41**:1, 1935.

24. Frank, Ira: The Deafness of Beethoven, Ann. Otol., Rhin. & Laryng. **44**: 327, 1935.

His death was finally attributed to cirrhosis of the liver. The deafness was first noticed when he was 26. Bone conduction was apparently good, as he used some sort of bone conduction device attached to his piano. At autopsy the auditory nerves were observed to be very much thinned and atrophied, but no proper examination of the middle and inner ears was made, although the temporal bones were removed. Subsequently, when Politzer wished to examine the specimens, they could not be found; so the pathogenesis of Beethoven's deafness will never be known. Frank is inclined to the view that Beethoven's deafness was due to otosclerosis.

TREATMENT OF INNER EAR DEAFNESS

Rudolf Panse [25] deplores the fact that it is impossible in cases of inner ear deafness to determine whether the lesion is in the labyrinth, the nerve or the brain. He regrets that pneumomassage has fallen into disrepute, as there is a distinct field for its employment. It improves the circulation in the middle and inner ears and in the auditory nerve. He supplies notes on twelve of his patients, all of whom showed improvement after treatment.

Yazujian [26] describes a new technic for applying diathermy in cases of chronic catarrhal deafness. One electrode is inserted into the lateral recess of the pharynx and the other into the external meatus. A headband with swivel joints is used for the ear electrodes. The nasopharyngeal electrodes are insulated, except for a roughened tip, around which cotton is wrapped and then dipped in saline solution. The nose and nasopharynx are swabbed with cocaine before the electrodes are inserted. The current is slowly turned on until there is a pleasant feeling of warmth. It is not desirable to go to the limit of tolerance. The treatment lasts for fifteen minutes and is given three times weekly. It is followed by pneumomassage. The author claims to have obtained good results in cases of chronic catarrh but not in cases of otosclerosis or chronic nerve deafness.

Sydney N. Parkinson [27] reports a case of "recurrent aural crisis," a term which he prefers to Ménière's syndrome, in which symptomatic improvement occurred after labyrinthine decompression by Portmann's technic. He discusses the views of Dandy, who has expressed the belief that the symptoms have nothing to do with the inner ear and are merely

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25. Panse, Rudolf: Behandlung des Innenohrleidens, Arch. f. Ohrenh-, Nasen- u. Kehlkopfh. **139**:244, 1935.

26. Yazujian, D. M.: Diathermy in Treatment of Chronic Deafness: New Technic, Laryngoscope **45**:230, 1935.

27. Parkinson, Sydney N.: Ménière's Symptom Complex: Endolymph Decompression with Symptomatic Improvement; Report of a Case, Ann. Otol., Rhin. & Laryng. **44**:382, 1935.

an explosion of function of the auditory nerve. The work of Portmann and more recently of Furstenberg and his colleagues has suggested that the dysfunction is in or at least includes the inner ear. Portmann in 1926 suggested opening the saccus endolymphaticus in such cases.

Parkinson reports the case of a dentist, aged 36, who was seen in 1932. He had suffered from recurrent attacks of vertigo, nausea and vomiting for over a year. Tinnitus and progressive loss of hearing in the left ear were noted. The crises had been occurring more frequently. A general examination revealed nothing abnormal. The cold caloric test produced nystagmus to both sides, but it was weaker on the left side and unaccompanied by nausea. In January 1933 the saccus endolymphaticus was exposed and divided. The postoperative course was uneventful, the patient returning to work in a week. There was immediate improvement in the tinnitus and vertigo. The nystagmus, nausea and vomiting disappeared. Two years later the patient was free from symptoms, but the hearing was reduced a further 20 per cent. There was less reaction to the caloric test on the left side.

Abstracts from Current Literature

Ear

CLINICAL ASPECTS OF DIPLACUSIS. G. KELEMEN, Monatschr. f. Ohrenh. **69**:722 (June) 1935.

Kelemen reports his observations on thirteen patients with diplacusis. He found that a small degree of musical sensitivity is sufficient to let the symptom become manifest. The diplacusis developed in association with the following disorders: acute inflammation of the middle ear, relapsing tubal catarrh, nonsuppurative exacerbations of residual processes, Ménière's disease and syphilitic neurolabyrinthitis, and it was found in some cases in which there were no other complaints. The author observed changes on the sound conduction apparatus in eleven of his thirteen patients. As the chief parallel symptoms he observed hyperacusis, poor hearing, tinnitus aurium and vertigo. He points out that although the character of the disturbance has been designated as harmonic, dysharmonic, echoic and so on a clear differentiation between these types is not always possible and it is not always possible to differentiate clearly between a monaural and a binaural diplacusis. The determination of the site of origin of the two elements of the diplacusis may make considerable difficulty. However, there are proofs that the disorder may originate at two sites within the same cochlea or on corresponding zones of the terminal apparatus of the two sides: at the periphery or in the supralabyrinthine region of the terminal apparatus, or at the periphery on the one and in a central location on the other side. Finally, there is the possibility that the condition is of purely central origin.

EDITOR'S ABSTRACT.

PATHOLOGY OF DEAFNESS CAUSED BY TUMORS OF THE MESENCEPHALON. H. BRUNNER, Monatschr. f. Ohrenh. **69**:970 (Aug.) 1935.

This paper is one of a series which Brunner publishes under the heading "Contributions to the Otologic Diagnosis of Tumors of the Brain." He reports the clinical history of one patient and describes the results of the necropsy. The petrous bones and the portion of the brain containing the tumor were subjected to microscopic studies. The changes in the left petrous bone differed from those in the right one in that the cochlear nerve in the internal auditory meatus was noticeably atrophic. Moreover, the organ of Corti had been changed into a row of flat cells, and the ganglion and nerve were absent. The macula sacculi was likewise somewhat atrophied. The tumor filled the caudal portion of the third ventricle and obstructed the passage of the aquaeductus cerebri into the third ventricle. The tumor reached almost to the floor of the third ventricle, and with a small process it reached into the aqueduct. In such a position, which explains the enormous dilatation of the lateral ventricles, the tumor exerted pressure on the adjoining portions of the brain stem, which were severely impaired thereby. Brunner describes the destructive action on the various cerebral structures and after summarizing his observations on the tumor points out that, on the basis of the clinical observations, it was not difficult to diagnose a tumor of the mesencephalon, and that the necropsy disclosed a pinealoma filling the caudal portion of the third ventricle. That auditory disturbances may occur in cases of pinealoma is well known. In this connection Brunner mentions that Bailey considers bilateral hardness of hearing as a frequent symptom of the presence of a pinealoma. Then Brunner makes comparisons with a case previously described by him in which the tumor was at practically the same site, that is, in the region of the acoustic isthmus, although it had originated on the brain stem rather than on the pineal

body as in the case under discussion. However, in both of these cases there developed a degeneration that descended to the organ of Corti in the cochlea and to the macula in the sacculus. To be sure, there were some differences between the two cases: In the case described here, the degenerative atrophy in the internal ear was much more severe than in the other case, and it was limited to the left side, whereas the atrophy in the other case had been bilateral. The explanation for this difference is seen in the fact that in the present case the acoustic isthmus was probably more severely impaired on the right than on the left side. Brunner admits that it is not yet definitely known which part of the acoustic isthmus has to be destroyed to effect an auditory disturbance, but he maintains that unilateral auditory disturbances (contralateral to the brain defect) do occur in cases of tumor of the mesencephalon.

EDITOR'S ABSTRACT.

IMPROVEMENT OF HEARING OF PATIENTS WITH DEFICIENT SOUND CONDUCTION BY MEANS OF HEARING APPARATUS UTILIZING BONE CONDUCTION OR AIR CONDUCTION. A. BRANDT, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:171, 1935.

Brandt describes his observations made on twenty-four patients. In twenty patients sound conduction was defective, and four had a complicated baryecoia. In eleven of the patients neither an apparatus that uses bone conduction nor one that utilizes air conduction improved the hearing; but in six patients hearing was improved by a bone conduction apparatus, and in six others the air conduction apparatus produced good results. In one patient both types of apparatus were effective. The author stresses the fact that in these experiments the patients did not have an opportunity to become accustomed to the hearing apparatus and that the apparatus might eventually be more effective than the first tests indicated.

EDITOR'S ABSTRACT.

CIRCUMSCRIBED MENINGITIS OF BROCA'S CENTER WITH SYMPTOMS OF ABSCESS OF THE TEMPORAL LOBE. A. JAUERNECK, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **26**:237, 1935.

Jauerneck describes the history of a man who had undergone a radical operation for the removal of a cholesteatoma that reached up to the temporal dura. On the eighth day following the operation he had an epileptiform seizure with clonic convulsions and an increase in temperature. After this a new intervention was made in which the dura was exposed. The dura was tautly stretched and had a protrusion in the center of which there was an area of granulation, but the suspected abscess of the temporal lobe could not be detected. Later there developed aphasia and paralysis of the right lower branch of the facial nerve. There were more epileptiform attacks, and the consciousness was impaired. There were clonic convulsions and cutaneous anesthesias, both of which involved chiefly the right side. Again an attempt was made to find an abscess of the temporal lobe but without success, and the patient died several days later. The brain showed hardly any signs of a diffuse meningitis, but above Broca's center for speech there was a round area, the size of a silver dollar, that was thickly covered with pus. Microscopic examination of this area disclosed a fibrinous purulent meningitis with deep-going cellular infiltration of the brain. The temporal lobes showed hardly any meningitic changes, and there was no temporal abscess. A short time later, the author observed a similar case: The patient was operated on for a cholesteatoma with a labyrinthine fistula. The temporal dura was tautly stretched. After six weeks the patient was discharged from the hospital. However, several weeks later he was admitted once more. He complained of severe headaches and pressure in the region of the temporal lobe. The cavity of the radical operation contained pus; the ear was deaf; the caloric irritability was greatly reduced, and sensory aphasia had developed. An abscess of the temporal lobe was searched for but without suc-

cess. The fever decreased, but the sensory aphasia remained, and motor aphasia, alexia and complete agraphia appeared. On the following day the temporal lobe was punctured once more, and an abscess was found. The patient recovered. The motor aphasia disappeared soon, but the sensory aphasia persisted for about four weeks. The agraphia disappeared after three weeks. Before resorting to the last puncture, it had been assumed that this patient might have a process similar to that detected in the first patient, and it was considered advisable to make a large trepanation in the temporal region where the focus was suspected. Whether such an intervention might have been successful in the first patient is doubtful. It remains unexplained why, in that patient, the process became localized at Broca's center, far removed from the primary focus.

Jauerneck considers such cases as rare but points out that Kuhn reported a similar case in 1897. To be sure, the meningitis was diffuse, but the process was especially severe in the region of Broca's center, and there were speech disturbances together with clonic convulsions on the right side, while the suppuration of the ear had been on the left side. This case had likewise been diagnosed as one of abscess of the temporal lobe.

EDITOR'S ABSTRACT.

Pharynx

PULMONARY INVOLVEMENT AFTER TONSILLECTOMY. W. SCHÜTZ, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:369, 1935.

Schütz states that Waldapfel, after his return from a trip to the United States, called attention to the higher incidence of pneumonia and of pulmonary abscesses after tonsillectomies in America as compared with central Europe. This and the fact that the etiology of postoperative pulmonary complications was still in dispute induced him to review 403 cases of tonsillectomy with regard to the postoperative pulmonary complications. He was able to corroborate Waldapfel's assertion that the pulmonary complications after tonsillectomies are less frequent than in America, for he found pulmonary complications in only thirteen of these cases. He sees the cause for this difference from the American incidence in the technic of the intervention, particularly in the fact that in his clinic nearly all tonsillectomies are made under local anesthesia and never under ether anesthesia. In analyzing the incidence of postoperative pulmonary complications he found that the largest number were caused by the hematogenic dissemination of small emboli that had been liberated by the operations, and he applies the term "interstitial pneumonia" to the pulmonary complications that are produced in this manner. In three of the cases the complications could be traced to aspiration. In discussing the diagnosis of pulmonary complications he stresses the importance of the roentgenologic examination.

EDITOR'S ABSTRACT.

TUBEROUS HYPERTROPHY OF THE PALATINE TONSILS. S. ZOLTÁN, Monatschr. f. Ohrenh. **69**:864 (July) 1935.

Zoltán cites a few cases from the literature in which nodules of the size of a pinhead or a pepper corn were found on the surface of hypertrophic tonsils, and then reports four cases of his own observation with the histologic changes. In evaluating his cases he emphasizes that his first case differed from the others in the histologic aspects. This case was one of a rare form of tuberculosis in which the productive process caused diffuse papillomatous hypertrophy of the surface of the tonsils. The histologic aspects in the other cases were different; on macroscopic inspection, the tonsils presented considerable similarity, but the histologic preparations showed differences. However, there was great hyperemia in all of them in spite of the fact that they appeared grayish-white. This apparent contradiction can be explained by the edematous perfusion of the tissues. The edema in the epithelium covered up the original color of the organs and gave them

an opaque appearance. Another common characteristic of the second group of cases was a deficiency in inflammatory processes. Most interesting was the behavior of the connective tissue. In some of the cases, the proliferation, loosening and hyaline sclerosis of the connective tissue dominated the histologic picture and were accompanied by atrophy of the lymph elements. Zoltán thinks that the apparent differences in the histologic aspects of the second group of cases can be explained by the differences in duration. He points out further that (according to one author) this second group includes also those cases in which hypertrophies develop on circumscribed parts of the tonsils, for instance, the so-called tonsillar polyps and accessory tonsils. In the last part of his report he makes suggestions regarding the possible course of the development and regarding the etiology of these tonsillar hypertrophies. EDITOR'S ABSTRACT.

OCCURRENCE OF STRIATED MUSCULATURE IN PALATINE TONSILS. L. HÖRBST and L. HASLHOFER, Monatschr. f. Ohrenh. **69**:927 (Aug.) 1935.

After citing other authors who observed striated muscle fibers in the tonsils, Hörbst and Haslhofer describe their own observations on fifty-one tonsils from twenty-eight children. When both tonsils were available usually one was sectioned parallel to the transverse axis. The authors observed striated musculature more or less far within the connective tissue septums of the tonsils from thirteen children. In most of these the musculature consisted of well preserved muscle bundles, oval on transverse section, whereas in several a few fibers lay loosely together, either stretched out or wavelike. The direction of these muscle tracts varied, for in the same section were sometimes both transverse and sloped ones. Although, in the majority, the musculature reached only to one fourth or to one third of the diameter of the tonsils, there were some in which the muscle bundles reached almost to the center. The authors show further that the connection of the intratonsillar muscular bundles with the musculature that adheres to the so-called tonsillar capsule cannot be denied. They call attention to connections between the base of the palatine tonsils and the pharyngopalatine and glossopalatine muscles, which in turn are girdled by fibers of the constrictor pharyngis; they point out that Glas saw in these connections of the palatine tonsils with the musculature of the constrictor pharyngis the cause for the appearance of muscular tissues in the tonsils. They show that this is the more understandable if the embryologic development of the tonsils is taken into consideration. They describe the embryologic development and reach the conclusion that in the arrangement and distribution of the muscular bundles there is a certain regularity, which is explained by the embryologic development of the tonsils. EDITOR'S ABSTRACT.

SHRINKING OF THE UVULA AFTER TONSILLECTOMY. K. BERNFELD, Monatschr. f. Ohrenh. **69**:932 (Aug.) 1935.

Bernfeld calls attention to a report on the fate of the uvula following tonsillectomy which was published by E. Fröschels *(Monatschr. f. Ohrenh.* **69**: 155 [Feb.] 1935) and then describes his own observations. In a study of about eight hundred uvulas he observed eight in which the shrinkage was severe, but he says that slighter changes in the volume were present. However, he observed not only shrinkage but, in two adults, swelling of the uvula. In discussing the causes of these changes he shows that the fate of the uvula is largely dependent on its involvement in the cicatrization after tonsillectomy. He stresses that among the patients showing severe uvular shrinkage he observed only one in whom the tonsillar niche was well preserved, whereas in the other seven he observed cicatrizations as well as more or less extensive obliteration of the tonsillar niche. Thus it appears that the shrinkage of the uvula can often be explained by mechanical factors in that the retracted uvula has been forced into the soft palate. As a second cause of the shrinkage, he considers the interruption of the lymph vessels

and the obliteration of the blood vessels, as suggested by Fröschels. However, he remarks that this obliteration is probably the result not so much of a changed postoperative distribution of fluid in the uvula as of cicatricial traction. He thinks that the enlargement of the uvula observed in the presence of cicatrization and obliteration of the tonsillar niches after tonsillectomy indicates that the latter changes usually reduce the lymphatic stream and also cause obstruction and stasis in the uvula. A third possible cause of the shrinkage of the uvula considered by Bernfeld is the probable reduction of the lymphoid elements in the uvula following tonsillectomy. EDITOR'S ABSTRACT.

ENDOTHELIOMAS OF THE PHARYNX. A. G. FETISSOV, Monatschr. f. Ohrenh. **69**:936 (Aug.) 1935.

Fetissov stresses that in the diagnosis of a tumor of the pharynx one should not overlook the fact that an endothelioma may develop in this region. He shows that opinions differ regarding the histogenesis and the histopathology of endotheliomas but that on the basis of present knowledge such growths may be considered to have a definite histologic structure and should be strictly differentiated, particularly from the so-called mixed tumors grouped with them. He emphasizes that endotheliomas develop from endothelium, which in turn develops from the mesenchyma of the embryonal connective tissue. Depending on the sites of origin, endotheliomas can be differentiated into angio-endotheliomas and endotheliomas that develop from the dura mater. The angio-endotheliomas can be subdivided into lymphangio-endotheliomas and hemangio-endotheliomas. The histologic structure of endotheliomas is characterized by polymorphism. In the same specimen there may be flat, cubic, longish and other forms of cells. The relationship between connective tissue stroma and parenchyma differs greatly in endotheliomas. In some tumors the one type of tissue predominates and in other tumors the other type. Another characteristic peculiarity of endotheliomas is their tendency toward degenerations of various types but particularly toward hyaline degeneration. In the discussion of the clinical course of endotheliomas Fetissov says that they may develop wherever there are blood and lymph vessels. They develop chiefly in the skin, the subcutaneous tissue, the cerebral meninges, the tissues of the oral cavity and those of the hard and soft palate. The development of such a growth in the pharynx is extremely rare. As regards the clinical course, endotheliomas stand at the dividing line between benign and malignant growths—those in which the connective tissue predominates are generally benign. In the beginning endotheliomas usually develop slowly, but later the rate of growth increases. Usually a capsule surrounds the growth. Endotheliomas do not metastasize, but they do recur occasionally. The prognosis is difficult because one cannot determine whether the growth will become malignant. Fetissov considers early surgical intervention the best treatment. After stressing once more that an endothelioma of the pharynx is extremely rare, he describes two that came under his observation in the course of fifteen years. Both growths were surgically removed. The tumors were the size of hen's eggs, but they caused only local disturbances; that is, they made swallowing rather difficult. The general condition of patients with pharyngeal endothelioma is usually not impaired. EDITOR'S ABSTRACT.

TOXAEMIA TONSILLOPULMONALIS FULMINANS INFANTUM. LORENTZ HEERUP, Hospitalstid. **78**:169 (Feb.) 1935.

By this name Heerup designates a violently acute infection that is frequently fatal within twenty-four hours, which may be sporadic or nosocomial, in poorly nourished children under 18 months old who often have had various catarrhal infections. Clinically, there are symptoms of septic hyperpyrexia without apparent focal involvement. On examination mild tonsillopharyngitis and obstructive hyperemic and edematous pulmonary involvement are found. Microscopically, a massive growth of staphylococci, streptococci and pneumococci appears in the

tonsils and lungs and frequently extends to the gastro-intestinal tract, but the blood and remaining organs are sterile. A theoretical explanation may be found in transmission from infant to infant, with increased virulence of the microbes, or in sensitization producing the pulmonary edema, with diffuse spreading of the microbes, just as in experiments on animals with pneumococci, and active absorption of the toxin in the tonsils and lungs.

VINTON, Brooklyn. [AM. J. DIS. CHILD.]

Larynx

CONGENITAL GLOSSO-EPIGLOTTIC CYST IN AN INFANT. ROBERT CLÉMENT and A. LEMARIEY, Bull. Soc. de pédiat. de Paris **31**:438 (Nov.) 1933.

A girl 2½ years old had been seen at the age of 3 months at the Trousseau Hospital because of severe respiratory difficulty. A month after birth she began to have stridor, which became progressively worse. There was also slight dysphagia. On admission to the hospital she had an infection of the upper respiratory tract, was markedly dyspneic, had cyanosis and appeared on the verge of suffocation. Roentgen examination showed no enlargement of the thymus or lymph glands and no opaque foreign body in the larynx, trachea or bronchi. Direct laryngoscopic examination revealed the presence of a cyst the size of a hazelnut in the right glosso-epiglottic fossa, pressing against the right half of the epiglottis and the right aryepiglottic fold and riding over the laryngeal cavity with every inspiration, almost blocking it. It was tense and translucent, and the surface appeared bluish, with a network of fine vessels over it. It was decided to aspirate this cystic tumor to relieve the dyspnea, and the external route was chosen because on a previous occasion when a similar growth was operated on through the mouth bronchopneumonia developed and the infant died. A needle was inserted through the integument of the neck; the tumor was pushed toward it by a finger introduced through the mouth, and from 2 to 3 cc. of thin serous fluid was withdrawn. Immediate relief from dyspnea was obtained, and the larynx regained a normal appearance. Contrary to expectation, the cyst did not refill, and two years later only a small fibrous nodule could be seen where the large swelling had been. It is thought that the cyst was probably congenital and that it was either of the dermoid type, related to the branchial hyoid arch, or belonged to tumors of the base of the tongue originating in the thyroglossal tract.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

RARE CASES OF INFREQUENT NONDIPHTHERITIC STENOSIS OF THE RESPIRATORY TRACT. HERMANN ZISCHINSKY, Jahrb. f. Kinderh. **144**:191, 1935.

Three cases of primary phlegmonous laryngitis are described. One was in a child of 7 years, another in a child of 19 months, and another in a child of 3½ years. All the children had noisy respiration, stridor and high fever. Following tracheotomy the inflammation subsided quickly, and the children were discharged cured. Another case in a child aged 2 years, with a fatal termination, is cited. The literature is reviewed, and the high mortality is stressed. A swollen epiglottis in the presence of the symptoms just described is suggestive of this condition.

A case of chronic hypertrophic laryngitis (verified by postmortem examination) in a mongolian idiot of 4½ years is described. Death followed the onset of symptoms comparatively suddenly. The early symptoms were undoubtedly unnoticed because of the low grade intelligence of the patient. Another case included in this category is that of a boy, 1 year of age, in which the early sign was a cough without laryngeal symptoms. Laryngeal symptoms appeared later. The duration of the illness was twelve weeks. Tracheotomy was necessary. Apparently a complete recovery followed.

Another striking case was that of a girl, 1½ years of age, who was ill for fourteen days. Symptoms of laryngeal stenosis appeared, which led to the thought

of diphtheria. A positive culture tended to confirm this suspicion. The course was rapidly down-hill, and autopsy revealed sepsis with mycotic aneurysm of the aorta, which compressed the left main bronchus and involved the left recurrent nerve.

The last case reported was that of a 4 year old girl with symptoms suggesting a foreign body, subglottic laryngitis or compression of the trachea due to glands. A roentgenogram showed tracheal compression due to two small objects in the immediate vicinity. The symptoms subsided, and within eight days the child was ready for discharge from the hospital. The etiologic factor was not determined, but the course suggested an infectious process.

LANDAU, Rochester, N. Y. [AM. J. DIS. CHILD.]

REFLECTED IMAGE OF THE ANATOMIC MECHANISM OF THE FALSE VOCAL CORDS IN RELATION TO THE RECURRENT NERVE. A. RÉTHI, Monatschr. f. Ohrenh. **69**:912 (Aug.) 1935.

This is another of a series of reports in which Réthi describes his studies on the innervation of the larynx. In summarizing the observations described in this report he says that in superficially anesthetized dogs unilateral cutting of the recurrent nerve is followed by a unilateral and bilateral cutting by a bilateral, paramedian position of the vocal cords. Regardless of whether it is unilateral or bilateral, the paramedian position is followed after a few days by an intermedian position. The appearance of the paramedian position is due to the auxiliary innervation. Réthi emphasizes that in human subjects unilateral paralysis is always followed by an intermedian position without it being possible to observe a transitional paramedian position as in dogs. In cases of bilateral paralysis there develops bilateral paramedian position, which in most cases is permanent. In human subjects as well as in dogs the position of the vocal cords after paralysis of the recurrent nerve is regulated chiefly by the transverse arytenoid muscle. Réthi compares the structure of this muscle in dogs and human subjects, and on this basis he explains the positions that result. He points out that, if in human subjects both recurrent nerves are paralyzed, the auxiliary innervation predominates and effects the bilateral paramedian position. He mentions and explains some exceptions to this rule. Further he describes experiments on dogs which demonstrate the rapidity of cure in paralysis of the recurrent nerve, and shows how this rapid cure was the cause of the erroneous conclusion that the resistance of the adductors is greater than that of the abductors. In cases of permanent paramedian fixation in human subjects, there develops first a contraction of the abductors, and later there follows shrinkage. If the healing is completed before the onset of the shrinkage, the vocal cord ceases to be in the paramedian position and makes normal movements. If the shrinkage precedes cure, the vocal cord remains in the paramedian position except that, during phonation, the median position may develop. If Réthi's operation is made on one side, the vocal cord which has been operated on remains in a lateral position and the rima glottidis is wide. In the event that the recurrent paralysis persists, the vocal cord which has not been operated on remains fixed during phonation; if the nerve functions again, this vocal cord swings past the median position over to the vocal cord that has been operated on.

EDITOR'S ABSTRACT.

Nose

IONIZATION OF THE NASAL MUCOSA: RELATIONSHIP BETWEEN REAGINS IN THE BLOOD AND EFFECT OF TREATMENT; PRELIMINARY REPORT. H. L. ALEXANDER and J. H. ALEXANDER, J. Allergy **6**:240 (March) 1935.

As the result of experimental work on fifty-three patients with nasal allergy on whom nasal ionization was done, twenty-five of whom were tested in two

weeks and in three months, the authors concluded that "it would appear that in patients seeking ionization treatment for nasal allergy the chances of relief are greater if no reagins in the blood are present. Whether or not this is a probability can be confirmed only by further statistics."

HOYER, Cincinnati. [AM. J. DIS. CHILD.]

THE FLOOR OF THE MAXILLARY ANTRUM. J. W. GERRIE, J. Am. Dent. A. **22**:731 (May) 1935.

In a comprehensive article dealing with the anatomy and pathology of the antrum Gerrie includes the following among his conclusions: Pathologic conditions in the upper alveolar process contribute the maxillary sinusitis in approximately 20 per cent of the patients with this condition. Alveolar abscess is seldom a demonstrable cause of maxillary sinusitis. Free socket dressings are dangerous in the presence of dental fistulas. Sinusitis following maxillary fracture is usually chronic. Soft organic foreign bodies more frequently cause acute suppurative sinusitis, while metallic ones more commonly cause chronic irritations. Gerrie believes that oral fistulas should not be used as a route for sinus irrigation. Reports of eight cases are included. NOYES, Chicago. [AM. J. DIS. CHILD.]

NASAL ORIGIN OF SEPTICEMIA IN THE NEW-BORN INFANT. ALBERTO MUGGIA, Pediatria **43**:202 (Feb.) 1935.

In further discussing the possibility that acute coryza in new-born infants may be complicated by sinusitis and pyemia, the author refers to his original report of 1909 and reports an additional case in a new-born infant whose infection was traced to an infected milk duct in the nursing mother. The author is of the opinion that such conditions are relatively frequent.

SIGNORELLI, New Orleans. [AM. J. DIS. CHILD.]

PLASTIC CORRECTION OF SADDLE NOSE. I. WALDE, Monatschr. f. Ohrenh. **69**:850 (July) 1935.

Walde estimates the efficacy of the various substances (autoplastic, heteroplastic and alloplastic) that are used for the correction of the saddle nose. He describes observations in ten cases in which a periosteum-covered splint from the tibia was used for the correction of the nasal deformity. In order to gain insight into the process of decomposition and attachment, the patients were from time to time subjected to roentgen examination. On the basis of his observations he contradicts Lexer, who assumed that the result at the end of three months is permanent. Walde found that between nine and eighteen months after implantation resorption ceases and ossification predominates, and he stresses that it is impossible to speak of a permanent result as long as resorption has not ceased. He concedes that the healing-in of the bone is an advantage of the autoplastic method, but he also calls attention to the disadvantages of the method. As such he considers: the necessity of an operative intervention to obtain the material for transplantation, the difficulty in shaping the material properly, and particularly the considerable shrinkage of the material and the hypercorrection necessitated thereby. He concludes that the splint which is to be implanted must be rather large and advises that a flap of periosteum should be applied to the side that is not covered with it. He considers the permanent cosmetic results of the implantation of a splint from the tibia as not entirely satisfactory but says that other autoplastic materials, such as a portion of the crest of the ilium, a part of a rib, cartilage with its perichondrium or fat tissue, give even less favorable permanent results. In discussing the use of heteroplastic material, he says that he tried the implantation of prepared cattle bone in two cases of saddle nose. In one patient the bone healed in, but in the other one it was expelled. Then he evaluates the

use of ivory and says that it does not heal in as readily as does autoplastic material, but that it surpasses the various alloplastic materials in this respect. It has the advantage that it can be shaped readily and that its resorption is slight. These two factors insure a favorable cosmetic effect. Moreover, the use of ivory does away with the necessity of an intervention to obtain the plastic material. He thinks that on the whole the use of ivory is as satisfactory as that of autoplastic material and that in some respects it is even more so. In regard to the alloplastic materials he says that, although many substances have been tried, none of them has given satisfactory results.

EDITOR'S ABSTRACT.

Miscellaneous

INTRACRANIAL COMPLICATIONS ARISING FROM BASAL FIBROID TUMORS. F. KOCH, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **139**:378, 1935.

Koch points out that basal fibroids may become manifest in various forms. These tumors occur almost exclusively in young men below the age of 25. The growth usually originates on the membrana basilaris of the occipital bone, but it may also develop on the periosteum of the first and second cervical vertebrae. The fact that basal fibroids develop only in young men may be related to the developmental peculiarity of the male cranium. In this connection he calls attention to the theory advanced by Bensch, according to which the periosteum of the cranial bones, for some unknown reason, is incapable of forming bony tissue and instead of becoming ossified becomes hypertrophic. Other authors, however, ascribe the development of basal fibroids to traumas, but the appearance of such tumors in the absence of traumas speaks against this theory. On macroscopic inspection, a basal fibroid appears as a nodular tumor of the nasopharynx. Finger-like proliferations of the tumor may penetrate into the adjoining accessory nasal sinuses, into the orbits, into the pterygopalatine fossa or even into the adjacent musculature. In exceptional cases the tumor may grow into the cranium, and intracranial complications may result. Quite frequently the tumor regresses, and in rare instances it is cast off. Koch describes the clinical history of a youth aged 18 in whom a basal fibroid had been diagnosed as a nasal polyp. The physician whom the patient consulted attempted to remove the growth but did not succeed. On the day after the intervention, severe chills, headaches and fever developed. The patient was hospitalized, and an operation was performed, but death followed. The necropsy revealed basal suppurating meningitis and other complications. The histologic examination of the tumor disclosed a basal fibroid. This case shows that after the development of intracranial complications the correct diagnosis of the original disorder may be extremely difficult. Moreover, it also demonstrates that every polypous growth of the nose should be treated by a specialist, and that, before an operation is decided on, one should determine whether cranial penetration has taken place. Intracranial growth usually contraindicates surgical removal because there is danger of injuring the dura. The danger of postoperative meningitis is especially great in removal of the lamina cribosa. Thus it is still a matter of dispute whether surgical or conservative treatment is best in cases of basal fibroid. What procedure to adopt has to be determined in each case.

EDITOR'S ABSTRACT.

CONSERVATIVE DIATHERMY IN OTORHINOLARYNGOLOGY AND RELATED FIELDS. H. SCHAEFER, Monatschr. f. Ohrenh. **69**:833 (July); 897 (August) 1935.

Following a discussion of the mode of action of conservative diathermy, Schaefer describes in detail the technic of its application, giving attention to the types of electrodes that are suitable for the ear, the nose, the tonsils and so on and to the strength of the current and the duration of the treatment. Further he reports and evaluates the therapeutic results he obtained in 152 patients whom he treated with conservative diathermy. On consideration of the severity of the

disturbances and of the possibilities of other therapeutic methods, he found that conservative diathermy produces favorable results in chronic otitis media, otosclerosis, chronic progressive labyrinthine baryecoia, ozena, chronic tonsillitis and chronic laryngitis. Other disorders in which conservative diathermy produced good results were external otitis, chronic catarrh of the middle ear and thyrotoxicosis. With the exception of ozena, the disorders belonging to the sphere of rhinology were not influenced. In some disorders in which constitutional factors play a part, conservative diathermy was combined with other therapeutic methods. Schaefer also mentions the contraindications to conservative diathermy. He concludes that, in view of the favorable results, it should be employed more widely in otorhinolaryngologic practice.

EDITOR'S ABSTRACT.

HYPOTENSION AND ITS RELATION TO BRAIN AND EAR. H. MAHLER, Monatschr. f. Ohrenh. **69**:952 (Aug.) 1935.

Mahler says that hypotension is observed chiefly in young persons. The most important clinical symptom of hypotension is the low blood pressure. The systolic blood pressure usually is between 90 and 100 mm. of mercury. The patients generally complain of fatigue, palpitation, somnolence, headaches and so on. Mahler gives the histories of eight patients who, in addition to the aforementioned general symptoms, had local disorders such as tinnitus aurium, vertigo and nystagmus; some also had migraine and psychic disturbances. Mahler thinks that these disorders are caused by circulatory disturbances in the auricular and cerebral spheres. For use in the treatment of the symptoms of hypotension he recommends substances that influence the vascular system, particularly epinephrine and related preparations.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Regular Monthly Meeting, Jan. 6, 1936

T. C. GALLOWAY, M.D., *President*

WALTER H. THEOBALD, M.D., *Secretary*

PHYSIOLOGY OF THE UPPER RESPIRATORY TRACT. DR. ANDERSON HILDING, Duluth, Minn. (by invitation).

This subject was chosen for discussion because it was specially requested.

For purposes of this paper, the functions of the nose are classified as primary and secondary. Primary functions are those which have to do with smelling and air conditioning; secondary functions are those which have to do with the disposal of the residue left by the inspired air and the protection of the mucous membrane from dangerous materials and pathogenic organisms which may be present in the residue. The primary functions are not discussed in this paper at any length. Practically the whole attention is given to the secondary functions.

Nowhere else in the body is such a delicate structure as the nasal mucosa so severely exposed to the environment. It would be quickly destroyed by adverse physical and chemical forces if it were not for certain special defenses which nature has set up. The following five lines of defense are discussed: (1) and (2) the chemical and physical defenses contained in the mucin film, (3) ciliary action, (4) the power of the epithelium to alter its form radically to meet the exigencies of circumstances and (5) the power of the epithelium to disintegrate and regenerate rapidly under irritation.

The film of mucus which covers all of the respiratory surfaces is considered to be the central factor in nasal physiology. At one and the same time it serves as a (1) humidifier, (2) protective blanket, (3) trap, (4) medium for ciliary action, (5) tenacious drag-net, (6) endless conveyer, (7) antiseptic, (8) chemical buffer, (9) diffusion medium, (10) lubricant and (11) diluent.

The chemical action of the mucin film is discussed in connection with the antiseptic lysozym and the hydrogen ion concentration. The physical characteristics of the mucin and their purposes are discussed.

The ciliary action is reviewed briefly, and a motion picture film to demonstrate this action is exhibited. The efficiency of ciliary action is illustrated by citing some of the recent experimental work done in connection with the study of silicosis.

The histologic characteristics of the respiratory epithelium as it is found in various locations are illustrated with slides, and the significance of its structural form and the relation of the latter to the force of the air flow is pointed out. Experimental work tending to show that the form of the epithelium can be radically altered by changing the air flow is cited.

The respiratory epithelium has the property of sloughing the living cells when it is subjected to an irritant such as the common cold; this is illustrated in a motion picture. The rapidity of repair following such sloughing is cited, and the need for more study of this property is pointed out.

DISCUSSION

DR. JOSEPH C. BECK: It certainly is interesting to see work like this again and again. I remember when Dr. Hilding first presented it at Atlantic City. I think that it is well to bring this work into the field of otolaryngologic practice,

and that the members may be perfectly logical in discussing it, particularly in regard to what has just been shown here: the making of windows in sinuses in the inferior meatus when really the action of the ciliary and mucous membrane is toward the middle meatus. I have always felt that a proper-sized opening, if one has to be made, should be made in the middle meatus. It is easier to make one there, and it may last about as long as some of the openings that are made in the inferior meatus, which rapidly close. I have had no personal experience with this procedure.

Another interesting thing in regard to the defense mechanism of the mucosa, in connection with the abuse the mucous membrane receives in treatment, particularly in that of sinusitis, has been shown in the work of the men who have demonstrated the influence of the various materials used. I think Dr. Hilding might speak of the use and abuse of lavage with all sorts of remedial agents.

Dr. Alfred Lewy: Dr. Beck has mentioned nearly all the clinical questions that occurred to me in regard to the movement of the cilia, but he did not enter into detail in regard to the effect of solutions used in treatment. All sorts of things are used—for instance, silver nitrate in solutions varying from 0.25 per cent strength to saturation—and particularly in hyperesthetic rhinitis are destructive agents being used, such as zinc in ionization. I do not know whether Dr. Hilding has gone into that subject or whether he believes that the regeneration which occurs in a few weeks is perfectly normal regeneration. I long ago gave up the application of caustic or irritating drugs to acute inflammatory lesions of the mucosa. I use milk of magnesia and olive oil, a therapeutic idea which I presented to this society some time ago.

An interesting observation in the pictures was that the movement of the cilia appeared as whorls. Were these always in the same direction? In the northern hemisphere whirlwinds are always contraclockwise, while in the southern hemisphere they are clockwise. I do not know the reason for this, but I wonder if the rule applies in Dr. Hilding's findings.

Dr. E. L. Ross: This presentation gives one a better idea of the delicate mechanism one deals with in treating the nose.

I wonder if Dr. Hilding has some further ideas on the therapy of the nose in addition to those suggested by Dr. Beck. The mucous blanket is broken up in acute rhinitis. There is a breaking down of the ciliated epithelium. There follows quick recovery. Consequently, must one save this mucous blanket by every means possible? Must one refrain from using drugs that will destroy or cause the ciliated cells to break off? Is it possible that these free cells do not carry with them organisms and that by this means the nasal cavity is not rid of infections?

Another thing that interests all: Why after the destruction of the surface epithelium does one see the cilia return and carry materials always in the same direction as before?

Dr. Francis L. Lederer: The members are always enlightened by a discussion of nasal physiology. The thought occurs to me that there is some contradiction in the evidence offered. Heine of Mosher's laboratory said that it was unnecessary to speak of these cilia as "poor cilia," which could be harmed by any such means as mentioned here tonight. He used upward of 11,200 roentgens and could not cause destruction of the cilia and concluded that ciliated epithelium is not a delicate structure, since it withstands these massive destructive rays with impunity. I realize that these cilia are important if Heine's studies are correct, but it seems to me difficult to believe that they respond to a 0.5 per cent concentration of silver nitrate to such a degree as has been mentioned or that zinc ionization has such a bad effect on nasal mucosa as some clinicians state. It would be interesting to know whether these cilia are so very susceptible as one is asked by some to believe and, on the other hand, are so resistant to massive doses of radiation.

Dr. Walter H. Theobald: I wish to thank Dr. Hilding for his excellent presentation. The clinical observation which I have made in reference to the movement of dyes on the living mucosa, which Dr. Hilding spoke of, I should

like to report briefly. The observation was first made by King, who used iodized poppy-seed oil 40 per cent. He injected this into the antrum and then was able to show its subsequent movement. Some years ago I showed some films of an experiment in which I used barium sulfate. This was injected into antrums into which permanent openings had been made in the course of surgical interventions. The exposures were made thirty minutes after the injection, twelve hours after, twenty-four hours after and so on up to a week. In every case it was interesting to note that with this heavy solution the cilia moved the barium sulfate up to the natural ostium and avoided the artificial opening. I tried the same experiment in antrums in which no artificial opening was made and was able in each instance to follow the movement of the solution up to the antral opening.

Dr. Austin A. Hayden: I do not know whether Dr. Hilding has explained to Dr. Lewy's satisfaction whether these currents move to the rule of the north or the south hemisphere, but I do know that Dr. Hilding's presentation has been more clockwise than many I have heard. He has presented a wealth of material in a most interesting way.

One thing I wish to ask: In his mention of primary functions of the nose and nasal sinuses he did not mention the function of resonance, and I wish to know what he thinks about that.

I wish to call to the chairman's attention the presence of Dr. Smith of Spokane and to ask him to extend to Dr. Smith the privileges of the floor.

Dr. Carroll Lee Smith, Spokane, Wash.: Dr. Hilding was raised in my country, Dr. Galloway is one of my college mates, and I feel right at home. I enjoyed Dr. Hilding's paper very much.

I wish to mention one thing Dr. Beck spoke of and use myself as an illustration. From 1918 to 1920 I was a sufferer from sinus trouble and ear trouble. In 1920 I had a radical operation on the left antrum, which was the most involved. Later I came to Chicago and one of the distinguished otolaryngologists of this city performed the operation on the right side of which Dr. Beck spoke and also took off the anterior half of the middle turbinate. I have never had any trouble on the left side, but on the right side, where there is a large opening, I wish to state that if I do not wash the antrum out every two days there is a rancid discharge which is not pus. I am opposed to openings such as I had. Mucus drains down from the ethmoid, and unless the antrum is washed out it is very uncomfortable.

Dr. T. C. Galloway: Does serum come through the nasal mucosa, or is all the nasal discharge a secretion of the glands? In acute rhinorrhea is there any fluid from the blood, or is the discharge secretory fluid? In the treatments in which mild protein silver packs are used is there any protective effect?

The members have taken Dr. Hilding far afield from his presentation, but I know he is interested in all these pathologic problems.

Under what circumstances does serum or exudate come from the blood, aside from the secretion of glands? Is there a protective flooding and washing out by these secretions in acute rhinorrhea? Does the treatment with mild protein silver or similar medicaments imitate a natural mechanism?

Dr. Anderson Hilding: There are many questions for which the answers, now unknown, must be found before therapeutics can be put on a rational basis. Some one spoke of the effect of medicines on the nose and the delicacy of the cilia and asked if the cilia are easily destroyed. They are delicate and easily destroyed, but, as I tried to point out, they are by no means helpless on that account. If destroyed, they are replaced with astounding promptness. It does no particular harm if that film of mucus is washed off, because it is all going to be down in the stomach anyway within thirty minutes and will be replaced. Irritants probably do no permanent damage. In this northern part of the country, practitioners use cocaine in the patients' noses during the winter just as at other times of the year, and the patients go out into the cold with their nasal spaces opened widely and the cilia paralyzed, and nothing particular happens.

In regard to the antral windows, they do not close because of the position or because they are insufficiently large but because the surgeon leaves a surrounding annular ulcer at the completion of the operation. Such ulcers heal with stenosing annular scars which may form a complete diaphragm, closing the window. It is quite possible experimentally to build a wall across a sinus where there was none before simply by causing desquamation of an annular band around the inside of the sinus. I have made this experiment repeatedly. A window in a sinus may be made to stay open indefinitely, even though small, if the edges of the mucous membrane on both sides are brought close together over the margins of the window. From the standpoint of physiology, one would hesitate to recommend the making of a large window in the middle meatus, for that is where the most active cilia are located and where the drainage is most rapid. It is quite conceivable that such a surgical window might seriously interfere with natural drainage. The sinus might even become entirely closed.

Ciliary action in regenerated mucous membrane seems to be well coordinated. This is a most interesting fact. How it is accomplished, I have no idea.

I do not know of any extensive work on the hydrogen ion concentrations characteristic of pathologic conditions. It is only recently that there have been any studies on the normal concentrations. The p_H goes up in the common cold, but I think that is because of the loss of carbon dioxide.

Dr. Hayden asked whether I consider resonance as a primary function of the nose. The classification which I have used is not given as a comprehensive classification of all nasal functions. It is merely a convenient device for locating and orienting the subjects to be discussed. Nasal resonance belongs in another field.

I do not know whether or not serum ever comes through the mucosa. Such membranes are transparent. As to the flow which results when one puts mild protein silver packs into the nose, I believe that is merely a flow of mucus and is a defense reaction against the medication itself.

SMOKE ASPHYXIA. DR. GEORGE S. LIVINGSTON.

A study was made of three cases of asphyxia due to smoke, one of them fatal, from clinical and pathologic standpoints. Two factors in asphyxia were recognized in all the cases, operating simultaneously yet independently of each other. The first is carbon monoxide anoxemia. The mode of action of carbon monoxide is reviewed. There is a chemical combination of the gas with hemoglobin which impairs the respiratory function of the blood, but the reaction is reversible, and the blood soon returns to normal in the presence of sufficient oxygen. The chief damage in carbon monoxide anoxemia is to the brain, which suffers severe degenerative changes. These changes are not specific for this gas, however, and have been produced experimentally in dogs both with carbon monoxide and with atmospheres deficient in oxygen.

The second asphyxiant factor is stenosis of the air passages as a result of a severe membranous inflammation due to the local irritant action of smoke, the mechanism of which is described in this paper.

The treatment requires separate attention to the two causative factors. Carbon monoxide anoxemia requires, when severe, early administration of inhalations of carbon dioxide and oxygen. Membranous laryngotracheobronchitis calls for early tracheotomy followed by frequent prompt removal of obstructing plugs and crusts.

DISCUSSION

DR. T. C. GALLOWAY: I think that this is a very important paper, for the condition which Dr. Livingston has so well described must frequently be overlooked. As one hears this discussion one does not realize how difficult it may be to diagnose tracheal obstruction in the presence of carbon monoxide poisoning. Owing to the effect of the carbon monoxide there may be difficulty in diagnosing obstructive dyspnea. I examined a patient suffering from this condition with a bronchoscope, and the air passages had almost exactly the appearance one expects in an ulceromembranous tracheobronchitis. Aspiration is most important.

DR. SAMUEL J. PEARLMAN: This is an unusual presentation and I am very happy to have heard it. I congratulate the essayist on his fine handling of a most interesting subject.

DR. ROLAND D. RUSSELL: I think this is an excellent and interesting paper, and it covers so many phases of the question that there is nothing much left to say. In regard to the oxygen and carbon dioxide method of treating a patient for asphyxia, I think that is not the only method of treatment. Hoge has treated patients who had ceased to breathe by injecting oxygen directly into the blood stream.

DR. SHERMAN L. SHAPIRO: Were any chronic changes observed in the children in the way of laryngitis or pharyngitis which could be attributed to the accident? I have had occasion to see such changes in firemen overcome by smoke, and also in one rather unusual case in which a previously healthy person was exposed for some time to the fumes of pyrene. In the last instance there was present an extreme form of hypertrophic bilateral laryngitis involving chiefly the false cords.

There is one question that always puzzled me which Dr. Livingston has cleared up: I have often wondered why masks for counteracting the effects of smoke were not in more general use among firemen, but on hearing about the different types of smoke that are encountered I realize how impracticable this would be.

DR. A. H. ANDREWS JR.: One thing I wish to call attention to is the use of blood transfusion in the treatment of this poisoning. As Dr. Livingston pointed out, there is a marked reduction of oxyhemoglobin and a reduction of hemoglobin, and it is therefore valuable to introduce normal hemoglobin into the blood stream.

Recently there has appeared in the literature references to the intravenous use of methylthionine chloride U. S. P. (methylene blue), the rationale being that the drug replaces the carbon monoxide in the compound with hemoglobin to form a less stable hemoglobin with a faster return to normal. The studies of Bussabarger and others have failed to corroborate this.

In addition to the changes which Dr. Livingston has mentioned there are circulatory changes, especially hemorrhages in the brain and liver which result in permanent impairment of their functions.

DR. T. C. GALLOWAY: Is there an effect from the heat of the flame? I think that must occur in some fires—for instance, in the Iroquois Theater fire, in which a sheet of flame swept out over the audience. I think, in some cases at least, heat must be a factor.

DR. GEORGE S. LIVINGSTON: I wish to thank Dr. Galloway for his help in the bronchoscopic removal of obstructions in one of the patients.

None of these children showed external burns. From the postmortem observations one cannot differentiate between changes due to smoke and those due to heat. There was no fire in the room in which the children were trapped, and it is probable that heat was of little or no importance as a cause of damage to the air passages.

There were no sequelae in the children who recovered. They have normal voices and normal breathing. One can readily see from the postmortem specimen, however, that there could be residual changes in the air passages, and many patients have them.

I do not know to what extent gas masks are used by firemen. Several types have been designed by Henderson and Haggard and by the United States Bureau of Mines.

In regard to blood transfusions, it is quite feasible to remove some of the patient's carbon monoxide hemoglobin and to replace it with a donor's oxyhemoglobin, but it must be done very soon after the rescue, since within a few hours most of the carbon monoxide will already have been displaced by oxygen if the carbon dioxide-oxygen treatment is used. Rarely can donors be found and typed within an hour or two, and after five or six hours it is useless. As to the use

of methylthionine chloride, I can see no basis for it. In the blood stream, it forms methemoglobin, which is a stable compound that cannot be dissociated by oxygen and must, therefore, increase the anoxemia instead of relieving it.

Regular Monthly Meeting, Feb. 3, 1936

T. C. GALLOWAY, M.D., *President*

WALTER H. THEOBALD, M.D., *Secretary*

(PROGRAM PRESENTED BY THE DEPARTMENT OF OTO-LARYNGOLOGY OF THE UNIVERSITY OF ILLINOIS MEDICAL SCHOOL)

INFLUENCE OF VARIATIONS OF OXYGEN AND CARBON DIOXIDE TENSION IN THE INSPIRED AIR ON HEARING, NYSTAGMUS AND AFTER-IMAGES. DR. IRWIN G. SPIESMAN (by invitation).

It is generally known that in no other branch of physiologic research is it more difficult to apply the results obtained in animals to man than it is in that concerning the brain. This holds true particularly for the investigation of sensory functions, which become evident to the experimenter only indirectly through alterations in the motor behavior. For this reason, it seems desirable, as far as circumstances permit, to study the function of the sensory nervous system in the human being.

The natural approach is through a study of the sense organs under conditions that influence the excitability of the central nervous system. In the present study the influence of deficiency of oxygen and that of variations in the carbon dioxide tension in the inspired air were chosen, since these factors are of a generally recognized importance for the excitability of any living substance and since normal conditions are almost immediately restored after readmission of air, because of the rapidity of the gas exchange.

Cortical excitability was studied by determining quantitatively the auditory threshold with a Western Electric audiometer and by measuring the latent period of negative after-images under standard conditions. The present report is based on several hundred experiments on well trained subjects. In the experiments with deficiency of oxygen concentrations varying from 7.5 to 15.8 per cent were used. Most effective were those of 10 per cent and less oxygen. It was observed that with the influence of deficiency of oxygen depending on the concentration of oxygen in the gas mixture, the duration of the test period and the sensitivity of the subject there occurred a gradual decrease in hearing. After readmission of air the normal auditory threshold was gradually reached. It was observed, however, that in experiments in which the oxygen content was low, the after-effects were very long lasting, the hearing remaining diminished for several hours in spite of the fact that the subject inhaled air. Even the inhalation of 50 per cent oxygen air mixture did not materially enhance the recovery.

Similar results were obtained in a study of after-images. Here, too, it was found that under the influence of deficiency of oxygen the after-images became dimmer and the latent period was greatly increased. In a number of persons the effect was so great that no negative after-image was obtained during the period of anoxemia and after readmission of air the return was gradual.

The second group of experiments concerned the effect of carbon dioxide on auditory and visual processes. It was observed that this gas in a concentration above 4 per cent had effects similar to those described for deficiency of oxygen, the main difference being that the recovery was much quicker on readmission of air.

A last group of experiments concerned the influence of reduced carbon dioxide tension in the blood on auditory and visual processes. The subjects inhaled

maximally at a rate of 35 or more inspirations a minute for from two to six minutes. This method of hyperpnea is very effective and is regularly followed by a distinct period of apnea. The effects of hyperpnea on auditory and visual processes are similar to those described for deficiency of oxygen and increase of carbon dioxide. The hearing is regularly decreased, and a lengthening of the latent period of negative after-images occurs as well as a decrease in intensity of the negative after-images. As far as the after-effects are concerned, the experiments with hyperpnea take an intermediate position between those with deficiency of oxygen and those with increase of carbon dioxide.

In all three groups of experiments it was occasionally observed that after readmission of air the hearing not only returned to normal but showed even a supernormal phase during which it was better than in the control periods.

The same three factors—deficiency of oxygen, excess of carbon dioxide and hyperpnea—have been investigated also for their effects on the subcortical processes. For this study caloric nystagmus was chosen. Very weak stimuli were employed according to the method of Veits, so that no cortical symptoms such as dizziness occurred. There was a marked difference between the effects on the auditory and visual processes and those on nystagmus. Whereas the number of nystagmic movements was decreased after administration of carbon dioxide, it was distinctly increased after hyperpnea, the brain stem thereby showing effects similar to those obtained on the spinal cord in tendon reflexes. Moreover, it was found that very low concentrations of oxygen had to be used in order to bring about any effects on nystagmus. The same persons who showed regularly a distinct loss in hearing during and after a short period of oxygen lack did not show change in nystagmic movements unless lower concentrations of oxygen were used over a prolonged period. In the effective experiments the number of nystagmic movements was decreased. There were, however, no after-effects, since the control experiments carried out in thirty minute intervals yielded the same values as were obtained before the period of oxygen loss.

Summarizing, it may be said that deficiency of oxygen, excess of carbon dioxide and reduction of carbon dioxide in the blood due to hyperpnea bring about similar results in vision and audition. The most effective means of reducing the intensity of these processes is that of reducing the oxygen tension in the inspired air, since in these experiments even long lasting after-effects were observed. In contradistinction to this it was found that nystagmus is influenced very little by deficiency of oxygen and that even when there are indications of influence after-effects are absent. This illustrates for the human brain the fundamental difference in sensitivity to oxygen between the cortex and the brain stem. Moreover, it is found that, whereas excess and deficiency of carbon dioxide have similar effects on cortical processes (reduction in the hearing acuity and diminution in the intensity of after-images) they cause opposite changes in nystagmus.

A close analysis of these observations together with the fact that only subjects trained for a long period were used rules out changes in attention as a factor contributing to the alterations in hearing and vision. It is highly probable that in these experiments the site of the alterations in hearing and vision was the cortex, and that of the changes in nystagmus, the brain stem and not the sense organs. The main differences between the effects obtained on the cortex and those on the brain stem were the absence of after-effects when the latter was involved, the greater susceptibility of the cortex to loss of oxygen and the fact that hyperpnea and excess of carbon dioxide have similar effects on the cortex whereas they display opposite effects when the brain stem is involved. These differences may all be reduced to one cause—the greater effect on the cortex of deficiency of oxygen—for this factor plays a rôle also in the experiment with hyperpnea, since the effect of hyperpnea on nervous tissue is the resultant of two antagonistic factors: (1) the specific increase in excitability of the nervous tissue whenever the carbon dioxide tension in the blood is decreased in hyperpnea and (2) the vasoconstriction which diminishes the oxygen supply to the tissues. If the sensitivity of the nervous tissue to the

latter factor is great, as is that of the cortex, the vasoconstriction may outweigh the other factor and, if it does, there is a decrease in excitability. If, however, the sensitivity to oxygen is not great, as has been shown to be true for the brain stem, the specific effect of the decreased carbon dioxide tension on the nervous tissue must result in increased excitability.

DISCUSSION

DR. ERNST GELLHORN: The essential point of the experiments which Dr. Spiesman carried out is that they proved the reactivity of the brain stem to be different from that of the cortex. Anoxemia and hyperpnea had the same effect on hearing and vision but opposite effects on caloric nystagmus; i. e., the reactivity of the brain stem to deficiency of oxygen and to hyperpnea is the same as that of the spinal cord; one may refer to the work of King, Garrey and Bryan (*Am. J. Physiol.* **102**:305, 1932), and to that of Strughold and Jörg (*Ztschr. f. Biol.* **94**:150, 1933). There seems to be an essential difference between the cortex and the rest of the nervous system, and this is probably due to the greater sensitivity of the cortex to deficiency of oxygen.

Studies involving negative after-images and more recent investigations with regard to intensity of visual discrimination showed results similar to those on hearing. The work was carried out with the idea of using the sense organs as an avenue of approach to problems involving the central nervous system in man. My colleagues and I have been interested in carrying out the work on human material, for the transfer of results obtained in experiments on animals to man is never more difficult than when those results relate to the central nervous system. We believe that this work has shown a characteristic difference in reactivity between the brain stem and the cortex. Psychic reactions follow the same course as other cortical processes, as recent (unpublished) studies have shown.

Is one entitled to say that the effects of deficiency of oxygen, excess of carbon dioxide and hyperpnea really concern the cortex and brain stem and not the peripheral sense organs? So far as vision is concerned one is not ready to offer definite proof. As to the hearing and the vestibular apparatus the facts are definite. It has been shown by H. Davis and collaborators that after the blood supply to the cochlea has been shut off for a long time a sound can be transmitted to the cochlea and the organ of Corti functions normally. As to the vestibular apparatus, observations made by Mowrer prove that the action currents led off from the vestibular nerve disappear at the end of the period of stimulation whereas the nystagmus may persist for a long time. This indicates that the phenomena in both cases are of central origin.

MANAGEMENT OF LABYRINTHINE INFECTION. DR. SHERMAN L. SHAPIRO.

This paper deals with my personal experiences in treating labyrinthitis in the course of middle ear infection. The classification into circumscribed labyrinthitis, paralabyrinthitis, serous labyrinthitis, diffuse manifest suppurative labyrinthitis and latent suppurative labyrinthitis, made by Ruttin, is followed as probably the best from a practical standpoint. In the treatment of circumscribed labyrinthitis the usual procedure of a radical mastoidectomy only is followed; paralabyrinthitis occurs most often in acute infections and requires drainage of the offending focus; serous labyrinthitis is treated conservatively, operation on the mastoid being performed only on the occurrence of definite symptoms of mastoiditis or when there is evidence of progressive involvement of the inner ear; diffuse manifest suppurative labyrinthitis is treated along the lines suggested by Lund, with absolute rest in bed as long as the cell count on the spinal fluid is within normal limits; latent suppurative labyrinthitis is treated surgically only on the usual indications for a radical mastoidectomy, and the radical mastoidectomy alone is performed if the history indicates that the labyrinth has been dead for several months or years. The operation on the labyrinth is reserved for (1) cases in which the cell count

on the spinal fluid, in the presence of acute suppuration of the labyrinth, indicates beginning meningitis; (2) cases of acute suppurative labyrinthitis or of a labyrinth recently dead in which mastoidotomy is neccessary because of other complications, and (3) cases of latent suppurative labyrinthitis in which sequestration of the labyrinth is found at operation. An interesting case of facial paralysis and serous labyrinthitis occurring in an acute exacerbation of a chronic infection, which later changed to a suppurative labyrinthitis, is given in detail.

DISCUSSION

DR. BRUNO BLUMKLOTZ: In relation to Dr. Shapiro's paper I shall outline briefly the management of labyrinthitis in von Eicken's clinic as I had the opportunity to observe it during a period of over four years.

I shall speak especially of diffuse labyrinthitis, which seems to be the point in dispute. In manifest labyrinthitis, serous or suppurative, if there was no indication for an operation on the mastoid the patient was carefully observed. If the loss of function in the inner ear progressed or there was an indication for a mastoidotomy the latter alone was performed when the spinal fluid was normal. If the cells in the spinal fluid were increased in number operation on the labyrinth was in order. In cases of chronic "dead labyrinth," if there was otherwise an indication for a radical mastoidal procedure, the latter only was done. I never saw meningitis follow such a procedure.

If a luxation of the stapes or an extraction of polypi or of foreign bodies was followed by acute suppurative labyrinthitis operation was resorted to immediately.

DR. ALFRED LEWY: This paper is full of dynamite, and there is not time to discuss it adequately. I agree in the main with Dr. Shapiro's presentation of indications. I have seen in the last two years seven patients who had labyrinthitis associated with chronic suppuration, two each with fistula. They were all treated conservatively so far as the labyrinth was concerned: six to eight weeks' rest in bed and then the radical operation on the mastoid. Now they are all well, with dry ears.

Dr. Shapiro did not mention petrositis, which is now attracting considerable attention among otologists. Symptoms pointing to the labyrinth are often due to inflammation of the petrosa by contiguity. It must be realized that the petrosa is involved just as the mastoid is in every case of severe otitis media and there may be symptoms referable to the labyrinth without actual invasion thereof.

Dr. Shapiro said that in the cases he has observed the cell count on the spinal fluid was not increased. Of course, that is Lund's great indication. He depends on pleocytosis as an indication for operating on the labyrinth in the treatment of the acute condition.

About twenty years ago when Ruttin's monograph on labyrinthectomy appeared I analyzed his results and found that the mortality was 17 per cent. His patients were treated on a more radical basis than was advised by Dr. Shapiro tonight.

BRONCHOSCOPIC ASPECTS OF MASSIVE COLLAPSE. DR. PAUL H. HOLINGER (by invitation).

The problem of massive collapse of the lung, or postoperative atelectasis, is one which has been of increasing interest to the bronchoscopist since Jackson, Lee, Tucker and others first demonstrated, in 1925, the dramatic relief that can be given to patients by bronchoscopic aspiration of obstructing secretions.

That atelectasis differs from pneumonia was shown over one hundred years ago, but confusion has continued to exist in spite of the fact that failure to make the correct differential diagnosis may result in a grave error, since almost every step in the treatment of a patient with pneumonia is contraindicated in the management of postoperative collapse.

The fundamental etiologic factor is bronchial obstruction, although for many years the condition was thought to be due to diaphragmatic paralysis. Preoperative and postoperative administration of sedatives, the use of atropine, previous patho-

logic conditions in the chest, tight abdominal binders and guarded respiration due to pain all tend toward the formation and stasis of thick tenacious secretion, producing bronchial obstruction.

There is much discussion as to whether the type of anesthetic used plays a rôle. Apparently it does not.

The first symptoms usually occur within from twenty-four to forty-eight hours after operation. Thoracic pain, persistence of a somewhat productive cough, rapid and shallow respiration, restlessness, rise in temperature, air hunger and cyanosis characterize the onset and progress. The physical findings essentially are a shift of the heart and mediastinum to the affected side, dulness, limitation of motion and elevation of the diaphragm. The breath sounds depend on the amount of obstruction, and they change rapidly as coughing dislodges the secretion. The roentgen observations are of the greatest importance and are shown in the slides. The shift of the heart and mediastinum are demonstrated as well as the area of density and the high immobile diaphragm. Compensatory emphysema of the opposite lung may be present.

On introduction of the bronchoscope through the larynx one is impressed by the tremendous amount of the secretion lying in the trachea and both bronchi. By using an aspirating bronchoscope and an independent aspirator one can aspirate more of this thick, viscid, tenacious secretion in a minute or two than the patient can cough up in several days. It is composed mostly of fibrin, with few bacteria or pus cells, unless the bronchoscopy is done after symptoms have been present several days, in which case the aspirated material is purulent and foul-smelling, having been an excellent culture medium for bacteria. At this stage the mucous membrane of the tracheobronchial tree is intensely red and edematous and, as Jackson and Jackson have shown, a purulent bronchitis has developed which, if untreated, may go on to abscess of the lung or bronchiectasis or may terminate fatally owing to asphyxia.

The change in the general condition of the patient following bronchoscopic aspiration is often as striking as that following tracheotomy. The temperature and the pulse and respiration rates drop, and the physical and roentgen findings show a rapid return to normal. Should there be, however, a tendency to replugging, one need not hesitate to do repeated bronchoscopic aspirations.

One frequently hears that patients with massive collapse of the lung will get well without specific treatment. This is not always true. In any case, after the correct diagnosis has been made, sedatives should be withheld, the position of the patient changed repeatedly and carbon dioxide rather than oxygen used to stimulate the respiratory excursions. When these methods fail bronchoscopic aspiration will hasten convalescence and prevent the more destructive pulmonary complications for which the prognosis is much more grave.

(Two cases were presented to illustrate the etiologic factors, the physical and the roentgen findings as well as the bronchoscopic picture.)

DISCUSSION

Dr. Alfred Lewy: I should like to ask whether Dr. Holinger has ever tried, in the treatment of his patients with abscess of the lung or bronchiectasis, catheterization without the use of the bronchoscope, as devised by me and several other men. At least the main bronchi can be reached in this way. Of course the bronchoscope is necessary for localization of the lesion, but after that suction through the catheter can be used to good advantage.

Dr. E. W. Hagens: In one or two patients it seemed as though the larynx was wide open, with little reaction, and it was possible to use the bronchoscope with practically no anesthetic. I thought that perhaps the complication had arisen at the time of the operation because of the fact that the larynx was wide open and there was poor sensitization of the tissue. I wonder if Dr. Holinger found that in this type of patient the larynx is wide open.

Dr. George H. Woodruff, Joliet, Ill.: A lobar pneumonia of nine days' duration was complicated by atelectasis. The question as to the indication for bronchoscopy arose. However, the patient died within twenty-four hours. I should like the essayist's opinion.

Dr. Samuel Salinger: Dr. Holinger would do well to read this paper before a surgical society. I believe most of the surgeons are aware of the benefit of bronchoscopy in cases of postoperative atelectasis, but many of them are not. I cannot see any danger in using the bronchoscope. I have used it in several patients with atelectasis and have been able to pass it without moving them from their beds.

The wide open larynx Dr. Hagens spoke of I also have seen, but I cannot say whether this condition was due to the action of the local anesthetic or to the atelectasis. At any rate, the bronchoscopy was accomplished with little trouble. The results were good.

Dr. T. C. Galloway: How much effect (in postoperative atelectasis) is there from turning the patient on the opposite side and shaking or slapping the chest?

Dr. Paul H. Holinger: I think that a catheterization in the manner described by Dr. Lewy could be done, but the patient cannot breathe through the catheter as he can through the bronchoscope, nor can one see what conditions are present as one can bronchoscopically. Under direct vision one can pass aspirators quickly and certainly into the affected bronchial branches, using the curved aspirators for the posterior or superior divisions.

In regard to the wide open larynx which Dr. Hagens mentioned, that condition may be due to the air hunger which the patient experiences. Very little anesthetic is needed. One reason for the condition may be that the patients usually have had before and after operation large amounts of sedatives. I prefer to use only a small amount of local anesthetic, because it is desirable to have the patient cough.

The postpneumonia atelectasis is quite a problem because with pneumonia on one side and atelectasis on the other the patient is extremely sick, having so little lung tissue with which to breathe. I do not like to use a bronchoscope in a moribund patient, but if there is a possibility of reaerating an atelectatic lung bronchoscopic aspiration may be a life-saving procedure. One wonders about the etiologic aspects of the original pneumonia—was it a typical pneumonia or was there also an atelectasis in that portion of the lung?

Dr. Galloway mentioned the procedure of turning the patient with a collapsed lung on his normal side and slapping or pounding the wall of the chest on the affected side with the fist. This is an excellent method of dislodging the secretory plugs, but one serious objection must be kept in mind: Should there be a sudden release of the secretion with the patient in this position it will run down the opposite side and obstruct the only remaining patent bronchus, resulting in a bilateral atelectasis instead of the original one-sided collapse, and a sudden "exitus" of the patient.

A New Method for the Fixation and Preparation of Gross Specimens. Dr. J. A. Weidemann (by invitation).

This new and original method is adapted especially to the anatomy of the ear, nose and throat. It employs linoleum lacquer and is relatively inexpensive and simple. It enables one to use material from cadavers which the dissecting rooms are finished with. Soft structures are kept intact which would otherwise deteriorate on long exposure to the air. My associates and I have subjected these prepared specimens to cold and heat and have found them unaffected; they are also dustproof and water-proof. The prepared models can be handled readily, probed and studied without fear of breakage, which makes them excellent for teaching purposes. In addition, they lend themselves readily to coloring and shading.

The models for teaching purposes currently available at commercial houses make it very difficult to pass a cannula or probe or to demonstrate them to students without extreme care, since the soft parts are made of wax or some waxy composition which is easily broken.

At the University of Illinois, Dr. Francis Lederer, of the ear, nose and throat department, has been looking for a method by which we could fix and preserve these specimens and have them as readily accessible and easily handled permanent models. As a result we have in our laboratories some forty-five well preserved specimens already of one year's duration, which is indicative of the practicability of this process.

HEMIPLEGIA FOLLOWING LIGATION OF THE CAROTID ARTERY FOR PHARYNGEAL HEMORRHAGE. DR. HAROLD J. CAWTHORNE and DR. ERNEST A. BREDLAU (by invitation).

On Oct. 16, 1935, a previously healthy colored child aged 3 fell from a rocking-chair and struck his head on the concrete floor. He cried vigorously at the time but soon resumed his play. Later in the evening he was feverish and fretful.

On the following day the child had what was described as a "cold," and his parents noticed that there was a nontender swelling on the side of the neck, in the region of the angle of the jaw. The child remained in bed, and on the following day a physician was called. The temperature was 101 F. A diagnosis of mumps was made.

On October 20, the child was worse. That night three or four drops of blood were found on his pillow which were thought to have come from the right ear. On the following day pus was found in the right ear.

On October 23, one-half cup of bright red blood gushed from the child's mouth. A physician was called, who said it was from the "cold" and was nothing to be alarmed about. At about 11 p. m., the child had another hemorrhage. He was brought to the dispensary at the Research and Educational Hospital the following morning; while there he had another hemorrhage of about one teacup of bright red blood and was admitted to the hospital.

General examination revealed considerable pus in the canal of the right ear with a perforation in the posterior inferior quadrant of the drum, marked swelling and injection of the right tonsil and surrounding tissue, displacement of the uvula to the left and a large tender mass in the right side of the neck, in the region of the superior anterior and posterior cervical lymph nodes.

The laboratory findings were: hemoglobin, 6 Gm. per hundred cubic centimeters of blood; erythrocytes, 2,140,000 per cubic millimeter; leukocytes, 21,750, with 86 per cent polymorphonuclears. A lateral roentgenogram of the neck showed an increased shadow of prevertebral soft tissue, suggestive of a retropharyngeal abscess. The temperature was 100 F.

A needle was inserted into the mass in the pharynx and only blood obtained. The condition of the child was such that it was feared another hemorrhage would prove fatal; ligation of the right common carotid was advised.

On October 25, when the carotid sheath was opened, the internal jugular vein was large and pulsating. The common carotid artery was ligated below the bifurcation. Immediately the dilation of the internal jugular vein decreased somewhat, but the vein was still markedly distended. The jugular vein was also ligated. The pharyngeal mass was explored and only a small amount of blood obtained.

On October 30, five days after the operation, it was noticed that the patient appeared inattentive and drowsy. On November 3, nine days after the operation, complete left hemiplegia developed. Potassium iodide was administered with the hope of hastening resolution of the cerebral lesion.

On November 16, thirteen days after the onset of the hemiplegia, it was observed that there was some movement in the left side of the face, also slight flexion and extension of the left leg and some movement in the fingers of the left hand. Improvement continued, and the patient was dismissed on December 8. At this time he could walk, but there was some motor impairment of the left arm; movement was more from the shoulder than from the elbow. Seen from time to time in the dispensary, the child has continued to show improvement, and at present little remains of the paralyses.

DISCUSSION

DR. ERNEST A. BREDLAU: The case which Dr. Cawthorne has just described presents certain features and problems which I should like to discuss.

It was apparent that the hemorrhage had to be controlled at once. And the choice between ligating the external and ligating the common carotid artery had to be made. A ligation of the latter was undertaken because the extensive phlegmonous inflammation made exploration higher in the neck inadvisable. Furthermore, it was impossible to be certain that the internal carotid artery was not directly involved.

In 1933 Salinger and Pearlman exhaustively reviewed the subject of hemorrhage from peritonsillar and retropharyngeal abscesses. They found no agreement among authors as to the ideal point of ligation. In the literature, some authors recommend exploration and ligation of the bleeding vessel; others favor ligation of the internal carotid artery, and some feel that equally good results follow ligation of the common carotid artery. Since hemorrhage may persist or recur following high ligation, I favor the ligation of the common carotid artery in most cases. The urgent character of the symptoms in the present instance precluded either gradual ligation or the use of fascia lata as a ligature, which has been advocated to allow circulatory compensation and to prevent injury to the intima.

The tremendous size of the jugular vein and the pulsation in it made ligation of it appear to be of value following the ligation of the carotid artery. There are wide differences of opinion as to the necessity of ligation of the jugular vein. I believe that its appearance, especially its size, should determine the decision.

The final point of interest in our case was the left hemiplegia which developed on the ninth postoperative day and which now, three months later, has practically disappeared. It is apparent that since recovery has taken place circulation in the right cerebral hemisphere has been reestablished. Since the hemiplegia was delayed we are justified in assuming that it was due either to an ascending thrombus or to an embolus. I should like to raise the question as to the rôle of the infection of the primary lesion in the development of the hemiplegia, since following a ligation of the common carotid artery in a youth of 18 who did not show infection the postoperative course was uncomplicated.

The time of onset of hemiplegia is indicative of its cause; i. e., if immediate, it is due to sudden anemia; if delayed, it is due to edema, softening, thrombosis or embolism.

My opinion is that, all other things being equal, the age of the patient is the determining factor in the prognosis, the dangers of ligation of the carotid artery increasing markedly in the higher age brackets.

Ligation of the carotid artery is a life-saving procedure, and such complications as are encountered, especially those in older persons, cannot be allowed to influence one's decision when this procedure is indicated.

DR. SAMUEL SALINGER: The points brought out by the authors are in line with the experiences reported in the literature, and I can agree quite well with everything which they have said. When there is a fulminating hemorrhage one cannot stop short of ligating the common or the internal carotid artery if it is accessible. It is important to remember that in ligating the common carotid artery one should at the same time tie off the external carotid artery, because it is possible for hemorrhage to recur after the ligation of the common carotid artery due to back pressure from the external artery past the bifurcation and into the eroded internal artery.

Dr. Bredlau pointed out the higher mortality with increasing age, and the mortality is higher, but there are some patients in whom cerebral complications following ligation of carotid arteries are due not to age alone but to the fact that the circle of Willis is open owing to congenital absence of one of the communicating arteries. Persons have come to postmortem examination in whom this condition has been found, so if one encounters cerebral involvement in a child following ligation of a carotid artery it is certain that the child has an anomaly—an open circle of Willis.

DR. T. C. GALLOWAY: I have ligated the common carotid artery eight times, with one death. It is a dangerous procedure, but it must be done in some cases.

OSTIUM MAXILLARE: AN ANATOMIC STUDY OF ITS SURGICAL ACCESSIBILITY. DR. O. E. VAN ALYEA.

Writers disagree widely as to the accessibility of the ostium maxillare. Those who clinically practice sounding the opening maintain that they can pass a catheter through it successfully in from 80 to 85 per cent of their cases. Other writers, basing their conclusions on anatomic findings, hold that the ostium may be entered with ease and without injury to surrounding structures in no more than 25 per cent of the cases.

An investigation of the ostium maxillare was made during the past year on all available cadavers in the anatomic laboratory of the University of Illinois. The study included eighty-four adult heads and several fetal heads of various ages. The specimens were divided in the midsagittal plane; the middle turbinate bone was pushed out of place, and in many cases the outer wall of the antrum was removed to facilitate the examination of the ostium. The ostium was located in the anterior third of the infundibulum in eight specimens, in the middle third in nine and in the posterior third in fifty-seven, and in ten it was visible at the extreme posterior tip. The uncinate wall was high (about 4 mm.) in twenty-one specimens and low (less than 2 mm.) in four. The hiatus was too narrow in ten specimens to admit the passage of an ordinary cannula. A low overhanging bulla ethmoidalis was present in five, and in sixteen the middle turbinate bone was low and of sufficient bulk to render probing impossible or extremely difficult; twenty-one, or 25 per cent, had accessory apertures, one having two such openings bilaterally. The entire group was tabulated as to the surgical accessibility of the ostium maxillare; in nineteen, or 22.6 per cent, the ostium was deemed entirely inaccessible. Six of these had accessory openings which from a clinical standpoint lowered the number of cases in which catheterization was impossible to thirteen, or 15.5 per cent. In forty-five specimens entrance of a probe was regarded as difficult but possible, and eleven accessory openings in this group brought the total in which catheterization was probably impossible down to thirty-four, or 40.5 per cent. The total of those in which it was easy to probe the ostium was increased from twenty-one, or 25 per cent, to thirty-eight, or 45.2 per cent.

The number of those in which the ostium was easily accessible justifies a technic for this procedure. In at least 20 per cent the natural orifice was entirely inaccessible, and in those remaining it was to be reached only with difficulty or with destruction of tissue. In such cases a great deal depends on the operator's dexterity, experience, technic, familiarity with anatomy and persistence. Prolonged search for the ostium is not advised, for this adds to the congestion and offsets in a large measure any good which may come from irrigation. Failure to find the natural opening readily should eliminate this method of approach in the individual case, and the irrigation should be carried out through a puncture in the wall. The puncture may be made in the inferior or in the middle meatus with equal effectiveness.

DISCUSSION

DR. JOHN A. CAVANAUGH: This is an interesting paper with interesting specimens. I do not agree with Dr. Van Alyea in regard to the method of washing the antrum with the cannula. I use the Pierce cannula and direct it through the antral opening in most instances. The youngest patient I have treated in this way was 2½ years old, and I had no difficulty. When the trocar is used, the patient objects to a second treatment. In some cases, I do not put the cannula through the antral opening but through the nasal fontanel. This is not so severe, and the patient has less discomfort than when a trocar is used.

I have encountered one so-called accessory antrum, which was in reality a large posterior ethmoid cell dipping down back of the antrum, and I wonder

whether this might not have been the finding in the specimen Dr. Van Alyea mentioned.

It is important to have a thorough anatomic knowledge of this area; otherwise, failure may result in the attempt to wash the sinus.

I wish to congratulate Dr. Van Alyea on his splendid presentation.

DR. GEORGE T. JORDAN: I enjoyed this paper and appreciate the immense amount of work Dr. Van Alyea did.

In regard to using the antral opening, I do not see much advantage in doing so. I feel that better results can be obtained by making an opening beneath the inferior turbinate bone; it gives the added advantage of drainage by gravity, and cure of the patient follows quickly and without much trouble.

DR. HARRY L. POLLOCK: This subject has been discussed so often and there is such difference of opinion that I suppose every one here has a different point of view. I have tried to wash out the antrum by placing a cannula through the antral opening, with very poor success, and I have found that patients complain more of this than of a puncture made through the inferior meatus. If one cocainizes the tissues thoroughly there is little trouble and the patients are willing to come back for a second washing. I agree with Dr. Jordan about making an entrance through the middle meatus with a trocar. I think one does more damage by trying to direct the cannula through the antral opening than by making an opening through the inferior meatus, and my results from the use of this method have been more gratifying.

DR. ROBERT SONNENSCHEIN: This excellent paper is of especial interest to me because about twenty-eight years ago I had the privilege of being instructed in the washing out of the antrum through the middle meatus by Dr. Pierce. Dr. Walter Theobald later became his disciple in this matter. Experience and the views of others help to decide what one wishes to do. If it is more pleasant for the patient (but I fail to find any for whom it is), to be treated by puncture by way of the inferior meatus, that procedure is proper for that type of patient; I have found, however, that this treatment is dreaded by most patients even though I try to cocainize the parts as completely as possible. Rarely do I make entrance via the inferior meatus. I first try—with thorough cocainization and endeavor to produce no trauma—passing the cannula through the antral opening. Very often the antral opening is large and there is sufficient by-path for the fluid to escape even with the cannula in place. If the natural opening cannot be entered, the cannula is inserted into an accessory opening which is present in 25 per cent of the cases. When this second route is not available I puncture the membranous portion of the fontanel, keeping the cannula in close contact with the upper border of the inferior turbinate bone. I admit that when there is tenacious pus this plan of puncture is not as good as flushing the antrum by way of the inferior meatus, but my co-workers and I succeed in most cases by using the methods stated.

DR. FRANK J. NOVAK JR.: I want to venture the comment that a paper of this kind justifies one's coming out on a night like this. This applies with equal force to the other papers on the program, and I think the University of Illinois Medical School is to be congratulated on assembling this program and presenting it to us.

DR. WALTER H. THEOBALD: Dr. Van Alyea has given a very complete demonstration of the ostium maxillare, but I rise to defend the group he spoke of, who wash out the maxillary sinus by way of an antral opening in 80 per cent of their cases. I cannot deny his statistical evidence. He gives a percentage that cannot be discredited. Nevertheless, Myerson and many others, including myself, are willing to state that 80 per cent of antrums treated, perhaps more, are irrigated by way of the natural or the accessory opening. There is one rule I lay down, and that is never to wash the antrum by means of a cannula unless there is free movement and play of the cannula in that ostium. If this rule is not observed there is danger of puncturing the wall and there is not room for the return of

the mucous or purulent material. To explain why 80 per cent of treated antrums can be irrigated through the antral opening, I may say that twenty years ago Pierce made the statement that most antral infections were in those antrums in which the ostia were least accessible, but clinical experience has proved this to be otherwise. It is my belief that the largest number of antral infections occur in those antrums in which the ostia are more accessible. Therefore, the patients one sees are those whose antral openings are accessible or whose antrums show the accessory opening. Therefore, the clinical observations do not check with the anatomic studies as to the accessibility of the ostium.

DR. T. C. GALLOWAY: I previously punctured the inferior meatus and some reactions occurred. Then the use of the Pierce cannula was satisfactory for several years, but I became convinced that in many instances the cannula did not go through the ostium. Now the use of the sharp Yankauer needle seems to me most simple, painless and successful.

DR. SAMUEL SALINGER: One can generally tell the age of the discusser by the fixity of his convictions on a particular topic. I myself still have an open mind!

As to irrigating the antrum through the antral opening I think one should be candid and state truthfully that one irrigates a large percentage of antrums through the fontanel, for often it is impossible to know definitely whether the cannula has entered an ostium or has penetrated the thin membranous wall. This is particularly true in the light of the anatomic facts presented by Dr. Van Alyea.

DR. O. E. VAN ALYEA: Most of the discussion seems to be of a clinical nature. Consequently I am not qualified to answer this part of it as a rebuttal of my paper, which is on the purely anatomic phase. I did make some observations, however, and shall attempt to maintain my stand on those.

Dr. Cavanaugh mentioned a posterior opening as leading to a possible second antrum. In my specimen the openings are both in the middle meatus; the cavities are maxillary in genesis as well as in topography, and the observation must be regarded as one of double maxillary sinus. There are many descriptions in the literature of the posterior ethmoidal cell extending laterally to the maxilla.

I notice the discussers belong to three classes: those who use the cannula, those who puncture the inferior meatus and those who attempt to use the cannula and, failing, puncture the middle meatus. Dr. Theobald stated that he irrigates the antrum only when he has free play for the cannula. The average diameter of the ostium in a large series is about 4 or 5 mm. If he encounters some obstacle in the hiatus, a low bulla ethmoidalis or some other structures, he will find difficulty in getting free play of the cannula.

I agree with Dr. Salinger, who said that there is no way of knowing whether the cannula is in the antral ostium or in the membranous area. This is due to the fact that the nasal fontanel is covered only by two very thin layers of membrane which fill in the spaces between the bony processes. One might easily penetrate these membranes with the tip of the cannula and get a free play between two of these osseous structures.

REGISTRY IN OTOLARYNGOLOGICAL PATHOLOGY: ARMY MEDICAL MUSEUM. DR. FRANK J. NOVAK JR.

It has long been felt as a great deficiency in otolaryngology that there has been no registry. Ophthalmologists have conducted such a registry for fourteen years, with great success. This year, owing to the financial support of the Academy of Otolaryngology, it has been possible to establish such a registry. This consists of a depository for specimens, which are mounted, prepared and made available to otolaryngologists throughout the country. A complete set of pathologic specimens can be secured for study or research. This registry cannot function by itself but must depend on contributions of specimens from those interested throughout the country. It is not asking too much to request an

otolaryngologist to part with a portion of a gross specimen and donate it to the registry if he knows that in doing so he is furthering the study in his specialty. Full credit is given to each one who donates a specimen, and each one who does receives a well prepared microscopic slide and a careful description of the pathologic changes in the tissues involved and the histopathologic diagnosis. If he wishes to have more specimens they are available. Unless the members of the Chicago Laryngological Society and other societies throughout the country support this enterprise it will take many years before such sets will be available for distribution. When they are ready, if any doctor desires any kind of a set he can write to the curator of the museum, make a small deposit to insure return of the specimens and be supplied with the material he asks for.

I urge all of the members here to keep this in mind and when one has a good specimen send at least a part of it to the Army Medical Museum for the registry.

A group for the study of pathology has been active in Chicago for three years. This is a free association of ten men, some of them fairly well prepared in general pathology but none adequately prepared in special pathology. This group studies such specimens as are available from the various institutions. Twice a month they meet, and some one presents some phases of pathologic study to which he has devoted considerable attention. The general outcome is that the men are getting a good idea of the pathologic changes in various conditions. Dr. Beck and I have felt that other groups should be started, and we will be glad to assist in any possible way if any one is interested in starting such a project.

Book Reviews

Allergy of the Nose and Paranasal Sinuses. A Monograph on the Subject of Allergy as Related to Otolaryngology. By French K. Hansel, M.D., M.S. Price, $10. Pp. 820, with 58 illustrations and 3 colored plates. St. Louis: C. V. Mosby Company, 1936.

This is an excellent scientific monograph on an involved subject. There is a thorough approach on the basis of the anatomy, histology, physiology and pathology of the nose and paranasal sinuses, as well as theoretical considerations of anaphylaxis. The clinical material is based on wide experience.

Some of the subjects are controversial, and the views of persons on both sides are fairly given. Some may feel that too much emphasis has been placed on an allergic basis of certain diseases, such as hyperesthetic rhinitis.

The value of zinc ionization is discussed, as well as the value of skin tests, the history, and cytologic examination of nasal secretion, the taking of a blood count, determination of the basal metabolism and other tests.

The nasal symptoms of sneezing, blocking and watery discharge may be caused by such a variety of conditions that the rhinologist may ascribe them to polyps or hyperplastic ethmoiditis, and the allergist, to specific sensitization. Failure to consider both sides of the question undoubtedly accounts for the many disappointments in both the surgical and the specific management of these conditions. The opinion that nasal polyps and hyperplastic ethmoiditis are manifestations of allergy is more widely held than formerly and should result in better management of these conditions.

This volume should accomplish the goal set in the foreword: to acquaint the rhinologist with some of the principles of allergy, as well as to acquaint the allergist with some related nasal problems.

There are many quotations from the literature and an extensive bibliography.

Operative Eingriffe im Gebiete des Ohres, der Nase, des Halses. Nach eigenen Erfahrungen Dargestellt. By Dr. A. Lautenschläger. Price, paper, 45 marks; bound, 48.60 marks. Pp. 171, with 355 illustrations. Berlin: Julius Springer, 1936.

This is a volume of 171 pages, with 355 illustrations, for the most part in colors. The chief value of the book is that it presents results of Lautenschläger's own experiences in operations in the field of otolaryngology. This author has been recognized for a long time as one of the foremost surgeons in this special field. The text and the illustrations present the matter in a clear fashion.

The author does not encroach on borderline operations, such as operations on the thyroid and laryngectomy. He restricts his discussions to the field ordinarily encompassed by the specialist in otolaryngology.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Prof. Gunnar Holmgren, Karolinska Mediko Kirurgiska Institut, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143 Groningen, The Netherlands.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Robert F. Ridpath, 1737 Chestnut St., Philadelphia.
Secretary: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Place: Atlantic City, N. J.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
President-Elect: Dr. Frank E. Burch, 408 Peter St., St. Paul.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: New York. Time: Sept. 26-Oct. 3, 1936.

American Bronchoscopic Society

President: Dr. H. M. Taylor, 111 W. Adams St, Jacksonville, Florida.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.

American Laryngological Association

President: Dr. Burt R. Shurly, 62 W. Adams Ave., Detroit.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. D. C. Jarvis, Quarry Bank Bldg., Barre, Vt.
Southern.—Chairman: Dr. Charles D. Blassingame, P. & S. Bldg., Memphis, Tenn.
Middle.—Chairman: Dr. Alfred Lewy, 25 E. Washington St., Chicago.
Mid-Western: Dr. Frederick A. Figi, 1013 First St., S.W., Rochester, Minn.
Western: Chairman: Dr. David R. Higbee, 3245 Fourth St., San Diego, Calif.

American Otological Society

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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